

=> file hcaplus

FILE 'HCAPLUS' ENTERED AT 14:22:02 ON 25 MAR 1998
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FILE COVERS 1967 - 25 Mar 1998 VOL 128 ISS 13
 FILE LAST UPDATED: 25 Mar 1998 (980325/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

This file now supports REGISTRY for direct browsing and searching of all non-structural data from the REGISTRY file. Enter HELP FIRST for more information.

=> d que 117

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L1 (      482)SEA FILE=HCAPLUS ABB=ON (P OR PLASMODIUM) (W) (FLACIPARUM
      OR OVALE OR VIVAX OR MALARIAE) OR (T OR TREPONEMA) (W) FALL
      IDIUM
L2 (      5655)SEA FILE=HCAPLUS ABB=ON SMALLPOX (W) VIRUS OR (M OR MYCOBA
      CTERIUM) (W) TUBERCULOSIS OR ASCARIS (W) LUMBRICOIDES OR DERA
      TOPHYTE
L3 (      34262)SEA FILE=HCAPLUS ABB=ON HIV OR IMMUNODEFICIEN? OR IMMUNE
      (W) DEFICIEN?
L4 (      158209)SEA FILE=HCAPLUS ABB=ON (E OR ESCHERICHIA) (W) COLI OR HIS
      TOPLASMA (W) CAPSULATUM OR HISTOPLASINOSIS OR (B OR BORRELI
      A) (W) BURGDORFERI OR LIME? (W) DISEASE# OR TYPHOID OR NORWAL
      K (W) VIRUS OR ROTOVIRUS
L5 (      20489)SEA FILE=HCAPLUS ABB=ON (L3 OR L4) (5A) (INHIBIT? OR TREAT
      ? OR THU/RL)
L6 (      49)SEA FILE=HCAPLUS ABB=ON L5 AND (L1 OR L2)
L7 (      26)SEA FILE=HCAPLUS ABB=ON L6 AND (HUMAN# OR CHIMP? OR MICE
      OR PIG# OR MONKEY#)
L8 (      3)SEA FILE=HCAPLUS ABB=ON L6 AND PARASIT?
L9 (      26)SEA FILE=HCAPLUS ABB=ON L7 OR L8
L10 (      6)SEA FILE=BIOSIS ABB=ON MALARIOTHERAPY
L11 (      2)SEA FILE=BIOSIS ABB=ON MALARIO? (W) THERAP?
L12 (      0)SEA FILE=HCAPLUS ABB=ON L10 OR L11
L13      26 SEA FILE=HCAPLUS ABB=ON L9 OR L12
L14      4389 SEA FILE=HCAPLUS ABB=ON FALCIPARUM
L16      14 SEA FILE=HCAPLUS ABB=ON L14 AND L5
L17      38 SEA FILE=HCAPLUS ABB=ON L13 OR L16

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=> file wpids

FILE 'WPIDS' ENTERED AT 14:22:13 ON 25 MAR 1998
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FILE LAST UPDATED: 23 MAR 1998 <19980323/UP>
 >>> UPDATE WEEKS:
 MOST RECENT DERWENT WEEK 199812 <199812/DW>
 DERWENT WEEK FOR CHEMICAL CODING: 199807
 DERWENT WEEK FOR POLYMER INDEXING: 199809
 DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE.
 >>> D COST AND SET NOTICE DO NOT REFLECT SUBSCRIBER DISCOUNTS -
 SEE HELP COST FOR DETAILS <<<
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>>> CHANGES TO DWPI COVERAGE - SEE NEWS <<<

=> d que 130

L18 (482)SEA FILE=HCAPLUS ABB=ON (P OR PLASMODIUM) (W) (FLACIPARUM
OR OVALE OR VIVAX OR MALARIAE) OR (T OR TREPONEMA) (W) PALL
IDIUM
L19 (5655)SEA FILE=HCAPLUS ABB=ON SMALLPOX(W)VIRUS OR (M OR MYCOBA
CTERIUM) (W)TUBERCULOSIS OR ASCARIS(W)LUMBRICOIDES OR DERA
TOPHYTE
L20 (34262)SEA FILE=HCAPLUS ABB=ON HIV OR IMMUNODEFICIEN? OR IMMUNE
(W)DEFICIEN?
L21 (158209)SEA FILE=HCAPLUS ABB=ON (E OR ESCHERICHIA) (W)COLI OR HIS
TOPLASMA(W)CAPSULATUM OR HISTOPLASINOSIS OR (B OR BORRELI
A) (W)BURGDORFERI OR LIME?(W)DISEASE# OR TYPHOID OR NORWAL
K(W)VIRUS OR ROTOVIRUS
L22 (20489)SEA FILE=HCAPLUS ABB=ON (L20 OR L21) (5A) (INHIBIT? OR TRE
AT? OR THU/RL)
L23 (49)SEA FILE=HCAPLUS ABB=ON L22 AND (L18 OR L19)
L24 (26)SEA FILE=HCAPLUS ABB=ON L23 AND (HUMAN# OR CHIMP? OR MIC
E OR PIG# OR MONKEY#)
L25 (3)SEA FILE=HCAPLUS ABB=ON L23 AND PARASIT?
L26 (5)SEA FILE=WPIDS ABB=ON L24 OR L25
L27 (6)SEA FILE=BIOSIS ABB=ON MALARIOOTHERAPY
L28 (2)SEA FILE=BIOSIS ABB=ON MALARIO?(W)THERAP?
L29 (0)SEA FILE=WPIDS ABB=ON L27 OR L28
L30 5 SEA FILE=WPIDS ABB=ON L26 OR L29

=> file biosis

FILE 'BIOSIS' ENTERED AT 14:22:24 ON 25 MAR 1998
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 20 March 1998 (980320/ED)
CAS REGISTRY NUMBERS (R) LAST ADDED: 20 March 1998 (980320/UP)

=> d que 146

L31 (482)SEA FILE=HCAPLUS ABB=ON (P OR PLASMODIUM) (W) (FLACIPARUM
OR OVALE OR VIVAX OR MALARIAE) OR (T OR TREPONEMA) (W) PALL
IDIUM
L32 (5655)SEA FILE=HCAPLUS ABB=ON SMALLPOX(W)VIRUS OR (M OR MYCOBA
CTERIUM) (W)TUBERCULOSIS OR ASCARIS(W)LUMBRICOIDES OR DERA
TOPHYTE
L33 (34262)SEA FILE=HCAPLUS ABB=ON HIV OR IMMUNODEFICIEN? OR IMMUNE
(W)DEFICIEN?
L34 (158209)SEA FILE=HCAPLUS ABB=ON (E OR ESCHERICHIA) (W)COLI OR HIS
TOPLASMA(W)CAPSULATUM OR HISTOPLASINOSIS OR (B OR BORRELI
A) (W)BURGDORFERI OR LIME?(W)DISEASE# OR TYPHOID OR NORWAL
K(W)VIRUS OR ROTOVIRUS
L35 (20489)SEA FILE=HCAPLUS ABB=ON (L33 OR L34) (5A) (INHIBIT? OR TRE
AT? OR THU/RL)
L36 (49)SEA FILE=HCAPLUS ABB=ON L35 AND (L31 OR L32)
L37 (26)SEA FILE=HCAPLUS ABB=ON L36 AND (HUMAN# OR CHIMP? OR MIC
E OR PIG# OR MONKEY#)
L38 (3)SEA FILE=HCAPLUS ABB=ON L36 AND PARASIT?
L39 (95)SEA FILE=BIOSIS ABB=ON L37 OR L38
L40 (89)SEA FILE=BIOSIS ABB=ON L39 AND 86215/BC
L41 (1)SEA FILE=BIOSIS ABB=ON L40 AND PROTECT?

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L42 (10)SEA FILE=BIOSIS ABB=ON L40 AND INHIBIT?
 L43 (2)SEA FILE=BIOSIS ABB=ON L40 AND PARASIT?
 L44 (6)SEA FILE=BIOSIS ABB=ON MALAROTHERAPY
 L45 (2)SEA FILE=BIOSIS ABB=ON MALARIO?(W)THERAP?
 L46 19 SEA FILE=BIOSIS ABB=ON L41 OR L42 OR L43 OR L44 OR L45

=> file medline

FILE 'MEDLINE' ENTERED AT 14:22:36 ON 25 MAR 1998

FILE LAST UPDATED: 19 MAR 1998 (19980319/UP). FILE COVERS 1966 TO DATE.

THE MEDLINE FILE WAS RELOADED FEBRUARY 15, 1998, TO REFLECT THE ANNUAL MESH (MEDICAL SUBJECT HEADING) CHANGES. ENTER HELP RLOAD FOR DETAILS.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

=> d que 164

L47 (482)SEA FILE=HCAPLUS ABB=ON (P OR PLASMODIUM) (W) (FLACIPARUM OR OVALE OR VIVAX OR MALARIAE) OR (T OR TREPONEMA) (W) PALLIDIUM
 L48 (5655)SEA FILE=HCAPLUS ABB=ON SMALLPOX(W)VIRUS OR (M OR MYCOBACTERIUM) (W)TUBERCULOSIS OR ASCARIS(W)LUMBRICOIDES OR DERA TOPHYTE
 L49 (34262)SEA FILE=HCAPLUS ABB=ON HIV OR IMMUNODEFICIEN? OR IMMUNE (W)DEFICIEN?
 L50 (158209)SEA FILE=HCAPLUS ABB=ON (E OR ESCHERICHIA) (W)COLI OR HISTOPLASMA(W)CAPSULATUM OR HISTOPLASINOSIS OR (B OR BORRELI A) (W)BURGDORFERI OR LIME?(W)DISEASE# OR TYPHOID OR NORWALK(W)VIRUS OR ROTOVIRUS
 L51 (20489)SEA FILE=HCAPLUS ABB=ON (L49 OR L50) (5A) (INHIBIT? OR TRE AT? OR THU/RL)
 L52 (49)SEA FILE=HCAPLUS ABB=ON L51 AND (L47 OR L48)
 L53 (26)SEA FILE=HCAPLUS ABB=ON L52 AND (HUMAN# OR CHIMP? OR MIC E OR PIG# OR MONKEY#)
 L54 (3)SEA FILE=HCAPLUS ABB=ON L52 AND PARASIT?
 L55 (26)SEA FILE=HCAPLUS ABB=ON L53 OR L54
 L56 (6)SEA FILE=BIOSIS ABB=ON MALAROTHERAPY
 L57 (2)SEA FILE=BIOSIS ABB=ON MALARIO?(W)THERAP?
 L58 (0)SEA FILE=HCAPLUS ABB=ON L56 OR L57
 L59 (91)SEA FILE=MEDLINE ABB=ON L55 OR L58
 L60 (9402)SEA FILE=MEDLINE ABB=ON HYPERTHERMIA, INDUCED+NT/CT
 L61 (9)SEA FILE=MEDLINE ABB=ON L59 AND L60
 L62 (46315)SEA FILE=MEDLINE ABB=ON ACQUIRED IMMUNODEFICIENCY SYNDROME+NT/CT
 L63 (14)SEA FILE=MEDLINE ABB=ON L60 AND L62
 L64 22 SEA FILE=MEDLINE ABB=ON L61 OR L63

=> file embase

FILE 'EMBASE' ENTERED AT 14:22:55 ON 25 MAR 1998

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FILE COVERS 1974 TO 20 Mar 1998 (19980320/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d que 174

L49 (34262)SEA FILE=HCAPLUS ABB=ON HIV OR IMMUNODEFICIEN? OR IMMUNE (W)DEFICIEN?

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L50 (158209) SEA FILE=HCAPLUS ABB=ON (E OR ESCHERICHIA) (W) COLI OR HIS
 TOPLASMA (W) CAPSULATUM OR HISTOPLASINOSIS OR (B OR BORRELI
 A) (W) BURGDORFERI OR LIME? (W) DISEASE# OR TYPHOID OR NORWAL
 K (W) VIRUS OR ROTOVIRUS
 L66 8410 SEA FILE=EMBASE ABB=ON (P OR PLASMODIUM) (W) FALCIPARUM
 L67 224428 SEA FILE=EMBASE ABB=ON L49 OR L50
 L68 267 SEA FILE=EMBASE ABB=ON L66 AND L67
 L69 152 SEA FILE=EMBASE ABB=ON L68 AND PARASIT?
 L70 31 SEA FILE=EMBASE ABB=ON L68 AND TREAT?
 L71 10 SEA FILE=EMBASE ABB=ON MALARIO THERAPY
 L72 21 SEA FILE=EMBASE ABB=ON L70 AND (HUMAN/CT OR APE# OR CHIM
 P# OR PIG# OR MICE OR MONKEY#)
 L73 16 SEA FILE=EMBASE ABB=ON L69 AND L72
 L74 26 SEA FILE=EMBASE ABB=ON L71 OR L73

=> file aidsline

FILE 'AIDSLINE' ENTERED AT 14:23:15 ON 25 MAR 1998

FILE COVERS 1980 TO 13 MAR 1998 (19980313/ED)

Aidsline has been reloaded with 1998 MeSH headings. See HELP RLOAD
 for details.

This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> d que 186

L47 (482) SEA FILE=HCAPLUS ABB=ON (P OR PLASMODIUM) (W) (FLACIPARUM
 OR OVALE OR VIVAX OR MALARIAE) OR (T OR TREPONEMA) (W) PALL
 IDIUM
 L48 (5655) SEA FILE=HCAPLUS ABB=ON SMALLPOX (W) VIRUS OR (M OR MYCOBA
 CTERIUM) (W) TUBERCULOSIS OR ASCARIS (W) LUMBRICOIDES OR DERA
 TOPHYTE
 L75 1560 SEA FILE=AIDSLINE ABB=ON L47 OR L48
 L78 2 SEA FILE=AIDSLINE ABB=ON MALARIA (L) TU/CT
 L79 42 SEA FILE=AIDSLINE ABB=ON L75 AND PARASIT?
 L80 4 SEA FILE=AIDSLINE ABB=ON L79 AND TU/CT
 L86 6 SEA FILE=AIDSLINE ABB=ON L78 OR L80

=> file cancerlit

FILE 'CANCERLIT' ENTERED AT 14:23:28 ON 25 MAR 1998

FILE COVERS 1963 TO 12 Feb 1998 (19980212/ED)

Cancerlit has been reloaded with 1997 MeSH headings. See NEWS FILE
 and HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate
 substance identification.

The problem with incorrect information in the Document Type (DT) field
 has been corrected.

=> d que 190

L47 (482) SEA FILE=HCAPLUS ABB=ON (P OR PLASMODIUM) (W) (FLACIPARUM
 OR OVALE OR VIVAX OR MALARIAE) OR (T OR TREPONEMA) (W) PALL
 IDIUM
 L48 (5655) SEA FILE=HCAPLUS ABB=ON SMALLPOX (W) VIRUS OR (M OR MYCOBA
 CTERIUM) (W) TUBERCULOSIS OR ASCARIS (W) LUMBRICOIDES OR DERA
 TOPHYTE

L87 989 SEA FILE=CANCERLIT ABB=ON L47 OR L48
L88 115 SEA FILE=CANCERLIT ABB=ON L87 AND TU/CT
L89 4076 SEA FILE=CANCERLIT ABB=ON ADJUVANTS, IMMUNOLOGIC+NT/CT
L90 5 SEA FILE=CANCERLIT ABB=ON L88 AND L89

=> dup rem 117 130 146 164 174 186 190

FILE 'HCAPLUS' ENTERED AT 14:26:07 ON 25 MAR 1998
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FILE 'BIOSIS' ENTERED AT 14:26:07 ON 25 MAR 1998
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FILE 'MEDLINE' ENTERED AT 14:26:07 ON 25 MAR 1998

FILE 'EMBASE' ENTERED AT 14:26:07 ON 25 MAR 1998
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FILE 'AIDSLINE' ENTERED AT 14:26:07 ON 25 MAR 1998

FILE 'CANCERLIT' ENTERED AT 14:26:07 ON 25 MAR 1998

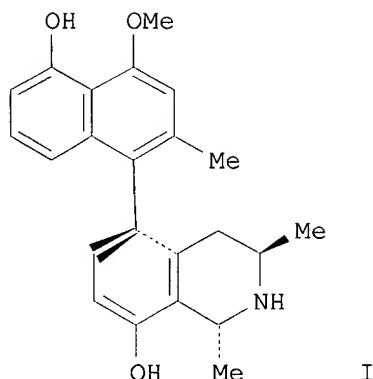
PROCESSING COMPLETED FOR L17
PROCESSING COMPLETED FOR L30
PROCESSING COMPLETED FOR L46
PROCESSING COMPLETED FOR L64
PROCESSING COMPLETED FOR L74
PROCESSING COMPLETED FOR L86
PROCESSING COMPLETED FOR L90

L94 108 DUP REM L17 L30 L46 L64 L74 L86 L90 (13 DUPLICATES REMOVED)

=> d 194 all 1-109



L94 ANSWER 1 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1998:20035 HCAPLUS
DN 128:154264
TI Acetogenic isoquinoline alkaloids. 105. Antiprotozoal activity of
naphthylisoquinoline alkaloids. 10. **HIV-inhibitory**
natural products. 44. First synthesis of the antimalarial
naphthylisoquinoline alkaloid dioncophylline C, and its unnatural
anti-HIV dimer, jozimine C
AU Bringmann, Gerhard; Holenz, Jorg; Weirich, Ralf; Rubenacker, Martin;
Funke, Christian; Boyd, Michael R.; Gulakowski, Robert J.; Francois,
Guido
CS Institut fur Organische Chemie, Universitat Wurzburg, Wurzburg,
D-97074, Germany
SO Tetrahedron (1998), 54(3/4), 497-512
CODEN: TETRAB; ISSN: 0040-4020
PB Elsevier Science Ltd.
DT Journal
LA English
CC 31-5 (Alkaloids)
Section cross-reference(s): 1
GI



- AB The first total synthesis of dioncophylline C (I), a new antimalarial lead structure, was described. For the directed construction of the stereogenic biaryl axis, the "lactone methodol." is applied, despite the lack of a "bridgehead oxygen" function in the target mol. The novel dimer of I, "jozimine C", was prepd., via oxidative phenolic coupling of the protected natural monomer. Jozimine C displayed good antimalarial activity (*Plasmodium falciparum*; IC₅₀ = 0.445 .mu.g/mL), and represents the first unnatural dimer of a naphthylisoquinoline alkaloid with a high anti-HIV activity (HIV-1; EC₅₀ = 27 .mu.g/mL).
- ST antimalarial naphthylisoquinoline alkaloid dioncophylline C prepn; jozimine C anti HIV dimer prepn; oxidative phenolic coupling jozimine dimer prepn; isoquinoline naphthyl alkaloid antimalarial prepn; dimer naphthylisoquinoline alkaloid anti HIV prepn; michellamine alkaloid prepn
- IT Antiviral agents
(HIV; prepn. of the antimalarial dioncophylline C and its unnatural anti-HIV dimer jozimine C naphthylisoquinoline alkaloids)
- IT Isoquinoline alkaloids
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation) (naphthyl; prepn. of the antimalarial dioncophylline C and its unnatural anti-HIV dimer jozimine C naphthylisoquinoline alkaloids)
- IT Antimalarials
Human immunodeficiency virus 1
(prepn. of the antimalarial dioncophylline C and its unnatural anti-HIV dimer jozimine C naphthylisoquinoline alkaloids)
- IT 146471-75-2P, (+)-Dioncophylline C
RL: BAC (Biological activity or effector, except adverse); RCT (Reactant); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(prepn. of the antimalarial dioncophylline C and its unnatural anti-HIV dimer jozimine C naphthylisoquinoline alkaloids)
- IT 202413-67-0P, (+)-Jozimine C
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(prepn. of the antimalarial dioncophylline C and its unnatural anti-HIV dimer jozimine C naphthylisoquinoline alkaloids)
- IT 162147-18-4 202215-71-2
RL: RCT (Reactant)
(prepn. of the antimalarial dioncophylline C and its unnatural anti-HIV dimer jozimine C naphthylisoquinoline alkaloids)
- IT 146471-72-9P 169168-95-0P 202215-64-3P 202215-66-5P
202215-67-6P 202215-69-8P 202215-73-4P 202215-75-6P

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202215-77-8P 202334-96-1P 202334-97-2P 202334-98-3P
202334-99-4P 202335-00-0P


RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. of the antimalarial dioncophylline C and its unnatural
anti-HIV dimer jozimine C naphthylisoquinoline alkaloids)

IT 202420-13-1P

RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of the antimalarial dioncophylline C and its unnatural
anti-HIV dimer jozimine C naphthylisoquinoline alkaloids)

L94 ANSWER 2 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1998:102258 HCAPLUS

TI Plasmodium **falciparum** antigen-induced human 
immunodeficiency virus type 1 replication is mediated through
induction of tumor necrosis factor-.alpha.

AU Xiao, Lihua; Owen, Sherry M.; Rudolph, Donna L.; Lal, Renu B.; Lal,
Altaf A.

CS Immunology Branch, National Center for Infectious Diseases, Centers
for Disease Control and Prevention, Division of Parasitic Diseases,
Atlanta, GA, USA

SO J. Infect. Dis. (1998), 177(2), 437-445

CODEN: JIDIAQ; ISSN: 0022-1899

PB University of Chicago Press

DT Journal

LA English

CC 15 (Immunochemistry)

AB Because malaria-stimulated cytokine prodn. may have deleterious
effects on human immunodeficiency virus type 1 (HIV-1) replication,
the effects of Plasmodium **falciparum** antigens on HIV-1
replication were studied. Stimulation with malarial antigens
significantly enhanced HIV-1 replication of HIV-1LAV and primary
HIV-1 isolates (subtype A) in CD8-depleted peripheral blood
mononuclear cells from naive donors. The malarial antigen-induced
activation of HIV-1 was due to cellular activation as judged by the
expression of cell activation markers and proliferative responses.
While malarial antigen stimulation increased expression of tumor
necrosis factor (TNF-.alpha.) and interleukin-6 (IL-6), only
monoclonal antibodies (MAbs) to TNF-.alpha. **inhibited**
malarial antigeninduced HIV-1 replication, whereas MAb to
IL-6 had no effect. Malarial antigen increased HIV-1 replication by
increasing viral mRNA expression and by activating long terminal
repeat-directed viral transcription. These data suggest that P.
falciparum infection can modulate HIV-1 pathogenesis by
activating lymphocytes and stimulating viral replication through the
prodn. of cytokines.

L94 ANSWER 3 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1998:27878 HCAPLUS

DN 128:153006

TI Mannan decelerates the clearance of human red blood cells in SCID
mouse

AU Ishihara, Chiaki; Hiratai, Rumi; Tsuji, Masayoshi; Yagi, Kazuaki;
Nose, Masao; Azuma, Ichiro

CS School of Veterinary Medicine, Rakuno Gakuen University, Ebetsu,
069, Japan

SO Immunopharmacology (1998), 38(3), 223-228

CODEN: IMMUDP; ISSN: 0162-3109

PB Elsevier Science B.V.

DT Journal

LA English

CC 15-8 (Immunochemistry)

AB Mannans and its related compds. decelerated human (Hu) red blood
cell (RBC)-clearance in severe combined **immunodeficiency**
(SCID) mice by **inhibiting** erythro-phagocytosis of

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macrophages. Chimeric SCID mice for Hu-RBC which are generated by repeated transfusions with mature Hu-RBCs are described recently as a model for Plasmodium **falciparum** infection, though the Hu-RBC clearance in the mice at present is very rapid and the parasitemia in the mice is only erratic. Here, we aimed to study the method to decelerate Hu-RBC clearance in SCID mice, to establish a suitable mouse model for malaria parasites. Yeast and Candida mannans as well as lactoferrin, a glycoprotein contg. both oligomannoside- and N-acetyllactosamine-type glycans, decelerated Hu-RBC clearance, but instead other saccharides such as carboxymethyl chitin, N-acetylglucosamine, and D--glucose did not. Yeast mannan and lactoferrin interfered significantly with in vitro Hu-RBC-phagocytosis which was also inhibited by mannopentaose and mannotriose. D-Mannose exhibited a moderate inhibitory activity. N-acetyl-D-glucosamine, however, showed only a slight inhibitory activity, but D--glucose had no inhibitory activity on Hu-RBC phagocytosis. These results may postulate that Hu-RBC clearance in SCID mouse might be mediated by receptor-ligand binding by a macrophage lectin like receptor with mannose specificity.

- ST malaria model erythrocyte phagocytosis macrophage mannan;
lactoferrin erythrocyte phagocytosis macrophage malaria model
- IT Biological simulation
Erythrocyte
Macrophage
Malaria
Mouse
Phagocytosis
Plasmodium **falciparum**
Severe combined immunodeficiency
(mannan, lactoferrin and D-mannose derivs. decelerate clearance
of human red blood cells in SCID mouse model for malaria)
- IT Lactoferrins
RL: BAC (Biological activity or effector, except adverse); BIOL
(Biological study)
(mannan, lactoferrin and D-mannose derivs. decelerate clearance
of human red blood cells in SCID mouse model for malaria)
- IT 3458-28-4, D-Mannose 9036-88-8, Mannan 28173-52-6, Mannotriose
70281-35-5, Mannopentaose
RL: BAC (Biological activity or effector, except adverse); BIOL
(Biological study)
(mannan, lactoferrin and D-mannose derivs. decelerate clearance
of human red blood cells in SCID mouse model for malaria)

L94 ANSWER 4 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS

AN 97:90270 BIOSIS

DN 99389473

TI Fever therapy: Lessons from the history and efficacy of
malariotherapy.

AU Stolley P D

CS Dep. Epidemiol. Preventive Med., Univ. Maryland Sch. Med., Baltimore,
MD 21201, USA

SO Mackowiak, P. A. (Ed.). Fever: Basic mechanisms and management,
Second edition. xix+506p. Lippincott-Raven Publishers: Philadelphia,
Pennsylvania, USA. 0 (0). 1997. 331-336. ISBN: 0-397-51715-7

DT Book

LA English

PR Biological Abstracts/RRM Vol. 049 Iss. 003 Ref. 036112

ST BOOK CHAPTER; TREPONEMA PALLIDUM; HUMAN; FEBRILE PATIENT; FEVER;
NEUROSYPHILIS; **MALARIOOTHERAPY**; PHARMACOLOGY; INFECTION;
NERVOUS SYSTEM DISEASE; BACTERIAL DISEASE; ANTIBACTERIAL
PHARMACOTHERAPY

CC Pathology, General and Miscellaneous-Therapy *12512

Nervous System-Pathology *20506

Pharmacology-Clinical Pharmacology *22005

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Pharmacology-Neuropharmacology *22024

Temperature: Its Measurement, Effects and Regulation-Thermopathology
*23007

Medical and Clinical Microbiology-Bacteriology *36002

Chemotherapy-Antibacterial Agents *38504

BC Spirochaetaceae 06112

Hominidae 86215

L94 ANSWER 5 OF 108 CANCERLIT

AN 1998031652 CANCERLIT

DN 98031652

TI Detection of bacillus Calmette-Guerin in the blood by the polymerase chain reaction method of treated bladder cancer patients.

AU Tuncer S; Tekin M I; Ozen H; Bilen C; Unal S; Remzi D

CS Department of Urology and Infectious Disease, Hacettepe University, Ankara, Turkey.

SO JOURNAL OF UROLOGY, (1997). Vol. 158, No. 6, pp. 2109-12.

Journal code: KC7. ISSN: 0022-5347.

DT Journal; Article; (JOURNAL ARTICLE)

FS MEDL; Cancer Journals; L; Priority Journals

LA English

OS MEDLINE 98031652

EM 199801

AB PURPOSE: Following intravesical bacillus Calmette-Guerin (BCG) instillation, we attempted to detect BCG in the blood using the polymerase chain reaction (PCR) method and correlate these findings with the occurrence of major complications due to this treatment. MATERIALS AND METHODS: Intravesical BCG immunotherapy was given to 22 consecutive patients with superficial bladder tumors. In 2 patients the BCG instillation had to be discontinued due to serious side effects of therapy. Blood samples (252 aliquots) were obtained from 126 BCG courses in 22 cases, and 2 additional samples (4 aliquots) were obtained from 1 patient 1 and 3 months after cessation of therapy. All blood samples were analyzed by the PCR technique for detection of deoxyribonucleic acid tuberculosis **Mycobacterium tuberculosis**. RESULTS: Of the 126 blood samples 9 (7.1%) were PCR positive for **M. tuberculosis**. These 9 positive samples belonged to 3 patients, all of whom were among those 4 patients who had major clinical side effects. CONCLUSIONS: We demonstrated that rapid and sensitive detection of mycobacteremia by PCR correlated with the clinical course of these patients. We also demonstrated that PCR can be used to monitor BCG in the blood after antituberculous therapy. The early, fast and accurate diagnosis of BCG in the blood by PCR may alter the serious clinical course of these patients by initiation of specific treatment early. However, further extensive studies are needed to validate these results.

CT Check Tags: Case Report; Female; Human; Male

*Adjuvants, Immunologic: BL, blood

Adjuvants, Immunologic: TU, therapeutic use
Aged

*BCG Vaccine: BL, blood

BCG Vaccine: TU, therapeutic use

*Bladder Neoplasms: BL, blood

Bladder Neoplasms: TH, therapy

Middle Age

Pilot Projects

*Polymerase Chain Reaction

CN 0 (Adjuvants, Immunologic); 0 (BCG Vaccine)

L94 ANSWER 6 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS

AN 97:516416 BIOSIS

DN 99815619

TI Does prior tuberculosis **protect human**

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immunodeficiency virus-infected persons from Mycobacterium avium complex disease? (and reply).

AU Collazos J

CS Section Infectious Diseases, Hosp. Galdakao, 48960 Vizcaya, Spain

SO Journal of Infectious Diseases 176 (5). 1997. 1412-1413. ISSN: 0022-1899

DT Short Communication

LA English

PR Biological Abstracts Vol. 104 Iss. 012 Ref. 173223

ST LETTER; MYCOBACTERIUM AVIUM; **MYCOBACTERIUM TUBERCULOSIS; HUMAN** IMMUNODEFICIENCY VIRUS; HIV; **HUMAN**; COMPLEX; PATHOGEN; PATIENT; TUBERCULOSIS; **HUMAN** IMMUNODEFICIENCY VIRUS INFECTION; HIV INFECTION; MYCOBACTERIUM AVIUM COMPLEX DISEASE; INFECTION; ANTIMYCOBACTERIAL IMMUNITY; RIFAMPIN; ANTIBACTERIAL-DRUG; ANTITUBERCULOSIS AGENT; ETHAMBUTOL; ANTIBACTERIAL-DRUG; AIDS; ACQUIRED **IMMUNODEFICIENCY** SYNDROME; IMMUNE SYSTEM; **TREATMENT** ; BACTERIAL DISEASE; VIRAL DISEASE; IMMUNE SYSTEM DISEASE

RN 74-55-5 (ETHAMBUTOL)
13292-46-1 (RIFAMPIN)

CC Biochemical Studies-General *10060
Pathology, General and Miscellaneous-Therapy *12512
Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System *15008
Pharmacology-Clinical Pharmacology *22005
Pharmacology-Blood and Hematopoietic Agents *22008
Pharmacology-Immunological Processes and Allergy *22018
Virology-Animal Host Viruses *33506
Immunology and Immunochemistry-Bacterial, Viral and Fungal *34504
Immunology and Immunochemistry-Immunopathology, Tissue Immunology *34508
Medical and Clinical Microbiology-Bacteriology *36002
Medical and Clinical Microbiology-Virology *36006
Chemotherapy-Antibacterial Agents *38504

BC Retroviridae 02623
Mycobacteriaceae 08881
Hominidae 86215

L94 ANSWER 7 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1997:337038 HCAPLUS

DN 127:12775

TI Retreatment tuberculosis cases Factors associated with drug resistance and adverse outcomes

AU Kritski, Afranio L.; De Jesus, Luis Sergio Rodrigues; Andrade, Monica K.; Werneck-Barroso, Eduardo; Vieira, Maria Armanda Monteiro S.; Haffner, Alice; Riley, Lee W.

CS Hospital Clementino Fraga Filho, Servico de Pneumologia, da Universidade Federal do Rio de Janeiro, Brazil

SO Chest (1997), 111(5), 1162-1167
CODEN: CHETBF; ISSN: 0012-3692

PB American College of Chest Physicians

DT Journal; General Review

LA English

CC 1-0 (Pharmacology)

AB A review with .apprx.16 refs. Risk factors assocd. with treatment failure and multi-drug-resistant tuberculosis (MDR-TB) were examd. among **HIV**-seroneg. patients who were previously **treated** for tuberculosis (TB). Prospective, cohort study of patients referred to the study hospital for retreatment of TB between Mar. 1986 and Mar. 1990. The patients belonged to three groups, according to outcomes following their previous treatment: 37 patients who abandoned treatment or suffered relapse after

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completion of therapy (group A), 91 patients who failed to respond to the first-line drug regimen (group B), and 78 patients who failed to respond to the second-line drug regimen (group C). Patients with **Mycobacterium tuberculosis** strains resistant to rifampin and isoniazid were found in 2 (6%) in group A, 29 (33%) in group B, and 49 (65%) in group C. Cure was achieved in 77% in group A, 54% in group B, and 36% in group C. Death occurred in none of the patients in group A, 8% in group B, and 24% in group C. In a multi-variate logistic regression anal., unfavorable response (failure to sterilize sputum culture, death, and abandonment) was significantly assocd. with infection with a multi-drug-resistant **M tuberculosis** strain ($p=0.0002$), cavitory disease ($p=0.0029$), or irregular use of medications ($p<0.0001$). These observations show that a previous treatment outcome and current clin. and epidemiol. histories can be used to predict the development of MDR-TB and adverse outcomes in patients undergoing retreatment for TB. Such information may be useful for identifying appropriate patient candidates for programs such as directly obsd. therapy.

ST review tuberculostatic drug resistance

IT Drug resistance

Tuberculostatics

(retreatment tuberculosis cases Factors assocd. with drug resistance and adverse outcomes in **humans**)

L94 ANSWER 8 OF 108 MEDLINE

AN 1998024480 MEDLINE

DN 98024480

TI Extracorporeal whole body hyperthermia treatments for HIV infection and AIDS.

AU Ash S R; Steinhart C R; Curfman M F; Gingrich C H; Sapir D A; Ash E L; Fausset J M; Yatvin M B

CS HemoCleanse Inc., West Lafayette, Indiana 47906, USA.

SO ASAIO JOURNAL, (1997 Sep-Oct) 43 (5) M830-8.

Journal code: BBH. ISSN: 1058-2916.

CY United States

DT (CLINICAL TRIAL)

Journal; Article; (JOURNAL ARTICLE)

(RANDOMIZED CONTROLLED TRIAL)

LA English

FS Priority Journals

EM 199803

EW 19980303

AB Whole body hyperthermia therapy (WBHT) is the elevation of the core body temperature to 42 degrees C. In vitro studies have confirmed that 42 degrees C is cytotoxic for virally infected lymphocytes, and even more effective when heating is repeated 4 days later. The safety and efficacy of two successive sessions of WBHT (4 days apart) was evaluated in 30 patients with AIDS (not on protease inhibitors), randomized to: 1) untreated controls, 2) low temperature WBHT for 1 hour at 40 degrees C and repeated 96 hours later, and 3) high temperature WBHT for 1 hour at 42 degrees C and repeated 96 hours later. The sorbent suspension in the ThermoChem System (HemoCleanse, West Lafayette, IN) system automatically controlled blood phosphate, calcium, and other electrolyte concentrations during WBHT. In 1 year of follow-up after WBHT, there were positive effects of the therapy on frequency of AIDS defining events, Karnofsky score, and weight maintenance. However, effects on plasma HIV RNA and CD4 counts were transient. Two successive WBHT treatments were performed in four patients who were on protease inhibitor/triple drug therapy, but had suboptimal response. In follow-up for 6 months, plasma HIV RNA and CD4 improved after WBHT, and the patients remained clinically well. This WBHT may have specific advantages in patients with suboptimal response to protease

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inhibitor therapy.

CT Check Tags: Human; In Vitro; Male; Support, Non-U.S. Gov't

Acquired Immunodeficiency Syndrome: PP, physiopathology

***Acquired Immunodeficiency Syndrome: TH, therapy**

Acquired Immunodeficiency Syndrome: VI, virology

Adult

CD4 Lymphocyte Count

Electrolytes: BL, blood

Extracorporeal Circulation: IS, instrumentation

***Extracorporeal Circulation: MT, methods**

Hemodynamics

Hyperthermia, Induced: IS, instrumentation

***Hyperthermia, Induced: MT, methods**

HIV Infections: PP, physiopathology

***HIV Infections: TH, therapy**

HIV Infections: VI, virology

Middle Age

RNA, Viral: BL, blood

CN 0 (Electrolytes); 0 (RNA, Viral)

L94 ANSWER 9 OF 108 AIDSLINE

AN 1997:20241 AIDSLINE

DN MED-97374346

TI Nitazoxanide in the treatment of cryptosporidial diarrhea and other intestinal **parasitic** infections associated with acquired immunodeficiency syndrome in tropical Africa.

AU Doumbo O; Rossignol J F; Pichard E; Traore H A; Dembele T M; Diakite M; Traore F; Diallo D A

CS Department of Parasitology, Mali National School of Medicine and Pharmacy, Bamako Mali.

NC N0125143

SO AMERICAN JOURNAL OF TROPICAL MEDICINE AND HYGIENE, (1997). Vol. 56, No. 6, pp. 637-9.

Journal code: 3ZQ. ISSN: 0002-9637.

CY United States

DT (CLINICAL TRIAL)

Journal; Article; (JOURNAL ARTICLE)

FS MED; Abridged Index Medicus Journals; Priority Journals

LA English

OS MEDLINE 97374346

EM 199710

AB Eighteen patients hospitalized with intestinal **parasitic** infections associated with diarrhea and dehydration completed a study of nitazoxanide in the treatment of *Cryptosporidium parvum* and other intestinal **parasitic** infections. Seventeen of the 18 patients were positive for human immunodeficiency virus. Twelve patients were diagnosed with clinical Stage 4 acquired immunodeficiency syndrome (AIDS) according to the 1990 World Health Organization proposed clinical classification system and cryptosporidiosis. Nitazoxanide (500 mg tablets) were administered orally, one tablet twice a day for seven consecutive days. *Cryptosporidium parvum* oocysts were eradicated or reduced by more than 95% in seven of the 12 Stage 4 AIDS patients who completed the study based upon two post-treatment fecal examinations conducted on days 7 and 14 following the initiation of treatment. The elimination or reduction of *C. parvum* oocysts was associated with a complete resolution of diarrhea in four of the seven patients. The test drug was also effective against cases of *Isospora belli*, *Entamoeba histolytica*, *Giardia lamblia*, **Ascaris lumbricoides**, *Enterobius vermicularis*, *Hymenolepis nana*, and *Dicrocoelium dentriticum*. Treatment with nitazoxanide was well tolerated by the patients. There were no abnormalities in blood chemistry or hematology data that were considered to be attributable to nitazoxanide therapy. Transient episodes of vomiting were observed

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in four patients, all with Stage 4 AIDS and cryptosporidiosis, which resolved spontaneously without discontinuation of treatment and were not considered to be related to administration of nitazoxanide.

CT Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

***Antiprotozoal Agents: TU, therapeutic use**

*AIDS-Related Opportunistic Infections: DT, drug therapy

Cryptosporidiosis: CO, complications

*Cryptosporidiosis: DT, drug therapy

Cryptosporidium parvum: DE, drug effects

Diarrhea: CO, complications

*Diarrhea: DT, drug therapy

Diarrhea: PS, parasitology

Intestinal Diseases, **Parasitic**: CO, complications

*Intestinal Diseases, **Parasitic**: DT, drug therapy

Mali

***Thiazoles: TU, therapeutic use**

RN 55981-09-4 (nitazoxanide)

CN 0 (Antiprotozoal Agents); 0 (Thiazoles)

L94 ANSWER 10 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.

AN 97383359 EMBASE

DN 1997383359

TI [Rapid tests for diagnosis of **parasitic** and fungal diseases].

TESTS RAPIDES POUR LE DIAGNOSTIC DES **PARASIToses** ET DES MYCOSES.

AU Robert R.

CS R. Robert, Laboratoire Parasitologie-Mycologie, Faculte de

Pharmacie, 16, boulevard Daviers, 49100 Angers, France

SO Immuno-Analyse et Biologie Specialisee, (1997) 12/5 (232-240).

Refs: 72

ISSN: 0923-2532 CODEN: IBSPEW

CY France

DT Journal; General Review

FS 004 Microbiology

026 Immunology, Serology and Transplantation

LA French

SL English; French

AB Today it is important to have good rapid methods for laboratory diagnosis of **parasitic** or fungal diseases. Indeed with modern high-speed travel and population movements, biologists anywhere may be called upon to diagnose cosmopolitan or tropical **parasitic** infections. Concerning mycosis, the incidence of opportunistic infections has increased progressively. They affect predominantly **immunodeficient** patients or patients under predisposing conditions (extensive surgical procedures or prolonged antibacterial, cytotoxic, or immunosuppressive **treatment**, among others). The definitive diagnosis of **parasitic** or fungal infections continues to be based on clinical criteria supported by procedures used in processing specimens for isolation and identification of **parasite** or fungi. The detection of the responsible agents is difficult in case of invasive infections. For this reason, immunological methods, used to detect soluble antigens or antibodies, were developed for the diagnosis of these diseases. Unfortunately, the classical methods (immunofluorescence assays, enzyme-linked immunosorbent assays, immunoprecipitation tests) are technically time consuming and specific therapy cannot be rapidly instituted. Therefore some rapid immunological tests for diagnosis of **parasitic** or fungal infections were developed. Commercialized latex agglutination tests are available for: diagnosis of toxoplasmosis and hepatic amoebiasis by antibodies detection; diagnosis of disseminated candidiasis, aspergillosis or cryptococcosis by antigens detection in serum, or cerebrospinal

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fluid; diagnosis of vaginal candidiasis by antigens detection in vaginal specimen; rapid identification of Candida colonies. Lateral flow immunochromatographic test sticks based on the detection of antigens in blood, serum or stool for diagnosis of **Plasmodium falciparum** infection, lymphatic filariasis or intestinal amoebiasis are marketed. These rapid tests are single step, sensitive and specific. They are easy to use and to interpret.

CT Medical Descriptors:

*parasitosis: DI, diagnosis
 *mycosis: DI, diagnosis
 *laboratory diagnosis
 antigen detection
 antibody detection
 immunofluorescence test
 enzyme linked immunosorbent assay
 immunoprecipitation
 latex agglutination test
 toxoplasmosis: DI, diagnosis
 liver amebiasis: DI, diagnosis
 candidiasis: DI, diagnosis
 aspergillosis: DI, diagnosis
 cryptococcosis: DI, diagnosis
 chromatography
 malaria falciparum: DI, diagnosis
 filariasis: DI, diagnosis
 amebiasis: DI, diagnosis
 human
 review

priority journal

Drug Descriptors:

*parasite antigen: EC, endogenous compound
 *parasite antibody: EC, endogenous compound
 *fungus antigen: EC, endogenous compound
 *fungus antibody: EC, endogenous compound

L94 ANSWER 11 OF 108 CANCERLIT

AN 1998041070 CANCERLIT

DN 98041070

TI Z-100, a polysaccharide-rich preparation extracted from the human type **Mycobacterium tuberculosis**, improves the resistance of Meth-A tumor-bearing mice to endogenous septic infection.

AU Sasaki H; Kobayashi M; Emori Y; Ohya O; Hayashi Y; Nomoto K
 CS Central Research Laboratories, Zeria Pharmaceutical Co., Ltd., Saitama, Japan.

SO BIOTHERAPY, (1997). Vol. 10, No. 2, pp. 139-43.

Journal code: AU3. ISSN: 0921-299X.

DT Journal; Article; (JOURNAL ARTICLE)

FS MEDL; L; Priority Journals

LA English

OS MEDLINE 98041070

EM 199802

AB The effect of Z-100, an immunomodulatory arabinomannan extracted from **Mycobacterium tuberculosis**, on cecal ligation and puncture (CLP)-induced sepsis in mice bearing Meth-A fibrosarcoma was investigated. When normal BALB/c mice were subjected to the CLP procedure, their mortality rate was 17%. On the other hand, an increased mortality was observed in tumor-bearing mice subjected to CLP 10 days after tumor inoculation, and then all mice died when tumor-bearing mice were subjected to CLP 20 days after tumor inoculation. However, the increased percent mortality was decreased by 50% when these mice were injected intraperitoneally with a 10 mg/kg dose of Z-100. When splenocytes (5 x 10⁷) cells),

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obtained from Meth-A tumor-bearing mice 20 days after tumor inoculation, were transferred intravenously to normal mice (recipient mice), mortality of these recipient mice were increased by 62% as compared with that of the control (22%). However, no increased mortality (25%) was observed in recipient mice which were transferred with splenocytes from tumor-bearing mice injected intraperitoneally with Z-100 (10 mg/kg). In addition, suppressor cell activity was demonstrated in splenocytes from Meth-A tumor-bearing mice at 20 days after tumor inoculation using one-way mixed lymphocyte reaction. However, the suppressor cell activity was significantly decreased by the intraperitoneal administration of a 10 mg/kg dose of Z-100 ($p < 0.01$). The increase of mortality in recipient mice by adoptive transfer of mononuclear cells (MNCs) from tumor-bearing mice was not detected when these MNCs were treated with anti-Thy 1.2 monoclonal antibody (mAb), anti-Lyt 2.2 mAb or anti-CD11b mAb, but an increase was seen with anti-Lyt 1.2 mAb or anti-immunoglobulin antiserum treated MNCs. These results suggest that the suppressor cells affect the mortality of CLP-induced sepsis and Z-100 may have a therapeutic activity against opportunistic infections in immunocompromised hosts through the regulation of suppressor T-cells.

CT Check Tags: Animal; Female; Male

***Adjuvants, Immunologic: TU, therapeutic use**

Cecum

Fibrosarcoma: CI, chemically induced

***Fibrosarcoma: IM, immunology**

Immunotherapy, Adoptive

Ligation

***Lipids: TU, therapeutic use**

***Mannans: TU, therapeutic use**

Mice

Mice, Inbred C57BL

Punctures

Sepsis: ET, etiology

***Sepsis: PC, prevention & control**

Spleen: CY, cytology

Spleen: DE, drug effects

Spleen: IM, immunology

T-Lymphocytes, Suppressor-Effector: DE, drug effects

T-Lymphocytes, Suppressor-Effector: IM, immunology

CN 0 (Adjuvants, Immunologic); 0 (Lipids); 0 (Mannans); 0 (SSM)

L94 ANSWER 12 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 1

AN 97:167297 BIOSIS

DN 99473900

TI **Malaria therapy** for HIV patients.

AU Heimlich H J; Chen X P; Xiao B Q; Liu S G; Lu Y H; Spletzer E G; Yao J L

CS Heimlich Inst., 2368 Victory Parkway, Suite 410, Cincinnati, OH, USA

SO Mechanisms of Ageing and Development 93 (1-3). 1997. 79-85. ISSN: 0047-6374

LA English

PR Biological Abstracts Vol. 103 Iss. 009 Ref. 129569

AB The objective of this study was to determine whether HIV patients who undergo **malaria therapy** experience beneficial immunological change without iatrogenic complications. In an approved, prospective study, asymptomatic, HIV-positive patients were inoculated with

P. vivax malaria and the malaria infection was

allowed to run a predetermined course according to standard

malaria therapy protocols and was cured with chloroquine.

After termination of the malaria, the patients have been followed for 2 years with clinical and immunological monitoring. In the first two HIV-positive patients, CD4 counts rose significantly from pre-malaria measurements and remain at normal levels 2 years later without

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AU Nasr, Mohamed E.
CS Division AIDS, National Institute Allergy and Infectious Diseases,
Bethesda, MD, 20852, USA
SO Book of Abstracts, 213th ACS National Meeting, San Francisco, April
13-17 (1997), CINF-023 Publisher: American Chemical Society,
Washington, D. C.
CODEN: 64AOAA
DT Conference; Meeting Abstract
LA English
AB The Division of AIDS (DAIDS) supports research to identify and
develop therapeutic agents for the prevention and **treatment**
of infections with the **human immunodeficiency**
virus (HIV) and assocd. opportunistic infections (OI's) including
Mycobacterium tuberculosis (TB). Computerized
data bases contg. chem. structures and biol. data have been
established by DAIDS that are designed to be the most up-to-date
information source on current research on HIV, OI's and TB exptl.
therapies. The data bases are currently managed using ISISBASE and
ISISHOST software of MDL Information Systems, Inc. The data bases
provide support for: (1) the acquisition, prioritization and to
avoid duplication of testing compds. for biol. evaluation in
contracts operated by DAIDS; (2) to track developments through
literature surveillance and abstraction of data on exptl.
chemotherapies of HIV and OI's; (3) to serve as knowledge base for
the NIAID and the scientific community; and (4) to prep. reviews on
structure activity relationships.

L94 ANSWER 15 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1997:116537 HCAPLUS
DN 126:122443
TI Vectors for the diagnosis and treatment of solid tumors including
melanoma
IN Pawelek, John M.; Bermudes, David; Low, Kenneth B.
PA Yale University, USA
SO PCT Int. Appl., 197 pp.
CODEN: PIXXD2
PI WO 9640238 A1 961219
DS W: AL, AM, AU, AZ, BB, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, IL,
IS, JP, KG, KP, KR, KZ, LK, LR, LS, LT, LV, MD, MG, MK, MN, MX,
NO, NZ, PL, RO, RU, SG, SI, SK, TJ, TM, TR, TT, UA, UZ, VN, AM,
AZ, BY
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB,
GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG

AI WO 96-US10250 960605
PRAI US 95-486422 950607
US 96-658034 960604
DT Patent
LA English
IC ICM A61K039-02
ICS A61K039-112; C07K014-525; C12N001-02; C12N015-63; C12N015-74;
G01N033-48
CC 63-3 (Pharmaceuticals)
Section cross-reference(s): 1, 16
AB The present invention is directed to the isolation and use of
super-infective, tumor-specific vectors that are strains of
parasites including, but not limited to, bacteria, fungi and
protists. In certain embodiments, the parasites include, but are
not limited to, the bacterium *Salmonella* spp., such as *Salmonella*
typhimurium, the bacterium *Mycobacterium avium* and the protozoan
Leishmania amazonensis. In other embodiments, the present invention
is concerned with the isolation of super-infective, tumor-specific,
suicide gene-contg. strains of parasites for use in treatment of
solid tumors.

ST antitumor microbial vector melanoma

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IT Antitumor agents
 Melanoma inhibitors
 (Vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Kidney tumors
 (inhibitors; vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Antitumor agents
 (kidney; vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Plasmids
 (pTK-Sec3; vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Genes (microbial)
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (suicide; vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Human herpesvirus
 (thymidine kinase gene of; vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Genes (microbial)
 RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); USES (Uses)
 (thymidine kinase-encoding; vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Chemotaxis
 (tumor-directed; vectors for the diagnosis and treatment of solid tumors including melanoma)

IT **Borrelia burgdorferi**
 Breast tumor inhibitors
 Brucella melitensis
 Chlamydia trachomatis
 Colon carcinoma inhibitors
 Cryptococcus neoformans
 DNA sequences
 Diagnosis
 Eimeria acervulina
 Encephalitozoon cuniculi
Escherichia coli
 Genetic engineering
 Hepatoma inhibitors
Histoplasma capsulatum
 Legionella pneumophila
 Leishmania amazonensis
 Leishmania major
 Leishmania mexicana
 Leptomonas karyophilus
 Listeria monocytogenes
 Lung tumor inhibitors
 Metastasis inhibitors
 Molecular cloning
 Mycoplasma hominis
 Neospora caninum
 Nosema helminthorum
 PCR (polymerase chain reaction)
 Phytomonas
 Plasmodium **falciparum**
 Pneumocystis carinii
 Prostatic tumor inhibitors
 Protein sequences
 Rochalimaea quintana
 Salmonella typhi
 Salmonella typhimurium

Sarcocystis suihominis
 Shigella
 Site-specific mutation
 Streptococcus
 Toxoplasma gondii
 Treponema pallidum
 Trypanosoma cruzi
 Unikaryon legeri
 Yersinia enterocolitica

(vectors for the diagnosis and **treatment** of solid tumors including melanoma)

IT Lipid A

RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Promoter (genetic element)

RL: BPN (Biosynthetic preparation); PEP (Physical, engineering or chemical process); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)
 (vectors for the diagnosis and treatment of solid tumors including melanoma)

IT Tumor necrosis factor .alpha.

RL: MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
 (vectors for the diagnosis and treatment of solid tumors including melanoma)

IT 82410-32-0, Ganciclovir

RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (vectors for the diagnosis and treatment of solid tumors including melanoma)

IT 9001-22-3, .beta.-Glucosidase 9001-45-0, .beta.-Glucuronidase
 9002-06-6, Thymidine kinase 9014-06-6, Penicillin V amidase
 9025-05-2, Cytosine deaminase 9037-41-6, Nitroreductase
 9055-15-6, Oxidoreductase 9073-60-3, .beta.-Lactamase 9074-87-7, Carboxypeptidase G2

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (vectors for the diagnosis and treatment of solid tumors including melanoma)

L94 ANSWER 16 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.

AN 96276487 EMBASE

TI Immunization of Aotus nancymai with recombinant C terminus of **Plasmodium falciparum** merozoite surface protein 1 in liposomes and alum adjuvant does not induce protection against a challenge infection.

AU Burghaus P.A.; Wellde B.T.; Hall T.; Richards R.L.; Egan A.F.; Riley E.M.; Ballou W.R.; Holder A.A.

CS Division of Parasitology, Ridgeway, Mill Hill, London NW7 1AA, United Kingdom

SO Infection and Immunity, (1996) 64/9 (3614-3619).

ISSN: 0019-9567 CODEN: INFIBR

CY United States

DT Journal

FS 004 Microbiology

026 Immunology, Serology and Transplantation

LA English

SL English

AB Merozoite surface protein 1 (MSP-1) of **Plasmodium**

falciparum is an antimalarial vaccine candidate. The highly conserved 19-kDa C-terminal processing fragment of MSP-1 (MSP-119) is of particular interest since it contains epitopes recognized by monoclonal antibodies which inhibit the invasion of erythrocytes in

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vitro. The presence of naturally acquired anti- MSP-119 antibodies in individuals exposed to malaria has been correlated with reduced morbidity, and immunization with an equivalent recombinant P. yoelii antigen induces substantial protection against this **parasite** in **mice**. We have expressed **P. falciparum** MSP-119 in **Escherichia coli** as a correctly folded protein and immunized Aotus nancymai **monkeys** by using the protein incorporated into liposomes and adsorbed to alum. After vaccination, the sera from these animals contained anti-MSP-119 antibodies, some of which competed for binding to MSP-119 with monoclonal antibodies that inhibit **parasite** invasion of erythrocytes in vitro. However, after challenge with either a homologous or a heterologous strain of **parasite**, all animals became **parasitemic** and required **treatment**. The immunization did not induce protection in this animal model.

CT EMTAGS: infection (0310); prevention (0165); therapy (0160); invertebrate (0723); protozoon (0751); genetic engineering and gene technology (0108); bacterium (0762); mammal (0738); nonhuman (0777); animal experiment (0112); animal model (0106); biological model (0502); article (0060); priority journal (0007)

Medical Descriptors:

*malaria: PC, prevention

*malaria: TH, therapy

*infection prevention

*active immunization

plasmodium falciparum

vaccine production

protein determination

immunogenicity

antigen recognition

expression vector

escherichia coli

aotus

nonhuman

animal experiment

animal model

article

priority journal

Drug Descriptors:

*malaria vaccine

*membrane protein

L94 ANSWER 17 OF 108 AIDSLINE

AN 1996:8685 AIDSLINE

DN MED-96261674

TI Circumventing genetic restriction of protection against malaria with multigene DNA immunization: CD8+ cell-, interferon gamma-, and nitric oxide-dependent immunity.

AU Doolan D L; Sedegah M; Hedstrom R C; Hobart P; Charoenvit Y; Hoffman S L

CS Malaria Program, Naval Medical Research Institute, Bethesda, Maryland 20889-5607, USA.

SO JOURNAL OF EXPERIMENTAL MEDICINE, (1996). Vol. 183, No. 4, pp. 1739-46.

Journal code: I2V. ISSN: 0022-1007.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

FS MED; Priority Journals; Cancer Journals

LA English

OS MEDLINE 96261674

EM 199610

AB Despite efforts to develop vaccines that protect against malaria by inducing CD8+ T cells that kill infected hepatocytes, no subunit

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vaccine has been shown to circumvent the genetic restriction inherent in this approach, and little is known about the interaction of subunit vaccine-induced immune effectors and infected hepatocytes. We now report that immunization with plasmid DNA encoding the plasmodium yoelii circumsporozoite protein protected one of five strains of mice against malaria (H-2d, 75%); a PyHEP17 DNA vaccine protected three of the five strains (H-2a, 71%; H-2k, 54%; H-2d, 26%); and the combination protected 82% of H-2a, 90% of H-2k, and 88% of H-2d mice. Protection was absolutely dependent on CD8+ T cells, INF-gamma, or nitric oxide. These data introduce a new target of protective preerythrocytic immune responses, PyHEP 17 and its P. falciparum homologue, and provide a realistic perspective on the opportunities and challenges inherent in developing malaria vaccines that target the infected hepatocyte.

CT Check Tags: Animal; Comparative Study; Female; Support, U.S. Gov't, Non-P.H.S.

CD8-Positive T-Lymphocytes: IM, immunology

*DNA, Protozoan: TU, therapeutic use

Genes, Protozoan

Immunity: GE, genetics

*Immunization

Interferon Type II

Lymphocyte Depletion

*Malaria: PC, prevention & control

***Malaria Vaccines: TU, therapeutic use**

Mice: GE, genetics

Nitric Oxide

Plasmids: TU, therapeutic use

Plasmodium yoelii: GE, genetics

Plasmodium yoelii: IM, immunology

Protozoan Proteins: GE, genetics

Protozoan Proteins: IM, immunology

Species Specificity

*Vaccines, Synthetic: TU, therapeutic use

RN 10102-43-9 (Nitric Oxide); 82115-62-6 (Interferon Type II)

CN 0 (circumsporozoite protein); 0 (DNA, Protozoan); 0 (Malaria Vaccines); 0 (Plasmids); 0 (Protozoan Proteins); 0 (Vaccines, Synthetic)

L94 ANSWER 18 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS

AN 96:521128 BIOSIS

DN 99243484

TI Pentoxifylline therapy in **human** immunodeficiency virus-seropositive persons with tuberculosis: A randomized, controlled trial.

AU Wallis R S; Nsubuga P; Whalen C; Mugerwa R D; Okwera A; Oette D; Jackson J B; Johnson J L; Ellner J J

CS Div. Infect. Dis., CWRU Sch. Med., BRB 1037, 10900 Euclid Ave., Cleveland, OH 44106-4984, USA

SO Journal of Infectious Diseases 174 (4). 1996. 727-733. ISSN: 0022-1899

LA English

PR Biological Abstracts Vol. 102 Iss. 011 Ref. 159114

AB Macrophage activation and tumor necrosis factor-alpha (TNF-alpha) production are critical in tuberculosis immunity but may result in increased **human** immunodeficiency virus (HIV) expression and accelerated HIV disease progression in **HIV**-infected persons. Pentoxifylline **inhibits** expression of TNF-alpha and **HIV**. A double-blind, placebo-controlled study of adjunctive therapy with pentoxifylline (1800 mg/day) as a timed-release formulation was done in Ugandan HIV-infected patients with pulmonary tuberculosis. Subjects had early HIV disease (mean CD4 cell count, 380/mu-L) and did not receive other antiretroviral drugs. Pentoxifylline resulted in decreased plasma HIV RNA and serum

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beta-2-microglobulin and, in a subset of moderately anemic patients, improved blood hemoglobin levels. Trends were noted toward reduced TNF-alpha production in vitro and improved performance scores, but these did not reach statistical significance. No effect was noted on body mass, CD4 cell count, or survival. Additional studies of more potent TNF-alpha **inhibitors** in **HIV-positive** subjects with tuberculosis are warranted.

ST RESEARCH ARTICLE; **MYCOBACTERIUM TUBERCULOSIS**;

HUMAN; **HUMAN IMMUNODEFICIENCY VIRUS**; **HOST**;

PATHOGEN; **INFECTION**; **PHARMACOLOGY**; **PENTOXIFYLLINE THERAPY**;

PENTOXIFYLLINE; **ENZYME INHIBITOR-DRUG**; **IMMUNOSUPPRESSANT-**

DRUG; **RANDOMIZED, CONTROLLED TRIAL**; **PHOSPHODIESTERASE**

INHIBITOR; **HUMAN IMMUNODEFICIENCY VIRUS**

INFECTION; **SEROPOSITIVITY**; **TUBERCULOSIS**; **TUMOR NECROSIS FACTOR-ALPHA**;

IMMUNE RESPONSE; **THERAPEUTIC METHOD**; **VIRAL DISEASE**; **BACTERIAL DISEASE**

RN 6493-05-6 (PENTOXIFYLLINE)

9025-82-5 (PHOSPHODIESTERASE)

CC Biochemical Studies-General 10060

Pathology, General and Miscellaneous-Therapy *12512

Pharmacology-Clinical Pharmacology *22005

Pharmacology-Immunological Processes and Allergy *22018

Immunology and Immunochemistry-Bacterial, Viral and Fungal *34504

Immunology and Immunochemistry-Immunopathology, Tissue Immunology *34508

Medical and Clinical Microbiology-Bacteriology *36002

Medical and Clinical Microbiology-Virology *36006

Chemotherapy-Antiviral Agents *38506

BC Retroviridae 02623

Mycobacteriaceae 08881

Hominidae 86215

L94 ANSWER 19 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.

AN 96212815 EMBASE

TI Photosensitized inactivation of **Plasmodium**

falciparum in human red cells by phthalocyanines.

AU Lustigman S.; Ben-Hur E.

CS New York Blood Center, 310 East 67th Street, New York, NY 10021, United States

SO Transfusion, (1996) 36/6 (543-546).

ISSN: 0041-1132 CODEN: TRANAT

CY United States

DT Journal

FS 004 Microbiology

025 Hematology

LA English

SL English

AB Background: Photodynamic **treatment** of red cell concentrate with phthalocyanines and red light inactivates lipid-enveloped viruses such as vesicular stomatitis virus and human **immunodeficiency** virus. This procedure is evaluated for its ability to enhance the viral safety of red cell concentrate for transfusion. It is of interest to study whether photodynamic **treatment** could also inactivate **parasites** in blood (e.g., **Plasmodium falciparum**). Study Design and Methods: Red cells **parasitized** by **P falciparum** were **treated** with phthalocyanines and red light and then cultured in vitro for 48 hours. The percentage of **parasitemia** was then estimated by microscopic examination of the red cells. Results: Of the phthalocyanines studied, the one that proved to be the most effective was $\text{HOSiPcOSi}(\text{CH}_3)_2\text{CH}_2)_3\text{N}(\text{CH}_3)_2$ (**Pc 4**). The extent of **parasite** inactivation increased with light dose and decreased with an increase in hematocrit. At a hematocrit of 60 percent and $2 \mu\text{M}$ **Pc 4**, $3 \log_{10}$ kill occurred at a light dose of 60 J per cm^2 . This is a lower dose than

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is required for .ltoreq.6 log10 of vesicular stomatitis virus inactivation (90 J/cm2). Conclusion: Photodynamic **treatment** with Pc 4 could make red cell concentrate not only virally safe for transfusion but also safe with respect to transmitting malaria.

CT EMTAGS: infection (0310); prevention (0165); therapy (0160); invertebrate (0723); protozoon (0751); virus (0761); mammal (0738); **human** (0888); controlled study (0197); human tissue, cells or cell components (0111); article (0060)
 Medical Descriptors:
 *malaria falciparum: PC, prevention
 *erythrocyte concentrate
 photodynamic therapy
 photosensitization
plasmodium falciparum
 infection prevention
 virus inactivation
 vesicular stomatitis virus
human
 controlled study
 human cell
 article
 Drug Descriptors:
 *phthalocyanine derivative

L94 ANSWER 20 OF 108 MEDLINE
 AN 97359908 MEDLINE
 DN 97359908
 TI Therapeutic hyperthermia in cancer and AIDS: an updated survey.
 AU Pontiggia P; Rotella G B; Sabato A; Curto F C
 CS Department of Hyperthermia and Oncology, Clinica Citt'a di Pavia, Italy.
 SO JOURNAL OF ENVIRONMENTAL PATHOLOGY, TOXICOLOGY AND ONCOLOGY, (1996) 15 (2-4) 289-97. Ref: 39
 Journal code: JOU. ISSN: 0731-8898.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LA English
 FS Priority Journals; Cancer Journals
 EM 199710
 EW 19971002
 AB The aim of this paper is to update with personal contributions the progress thus far accomplished in the clinical application of hyperthermia (HT) in cancer and chronic infectious diseases. The HT treatment has been successfully developed since the 1970s in cancer patients in whom it showed positive results consisting of complete or partial clinical remissions. Its rationale was based on the fact that core temperatures of > or = 42 degrees C induce cytotoxic effects that are higher in malignant cells than in normal cells. HT could be applied by different methods according to type, stage, and localization of the malignancies. Thus, systemic whole-body HT (WBH), through invasive or noninvasive techniques, was first used in disseminated cancers; local perfusion, infusion, and interstitial HTs have been applied in limb, skin, subcutaneous, or intracavitary tumors. The observation of a macrophagic lysosomal exocytosis and subsequent cancer cell death induced by HT, suggested that its mechanism of action involves an immune reaction. This suggested the possibility of associating HT with cytotoxic agents, antibiotics, antiviral drugs, and antioxidants, including beta-carotene (BC). The association of HT with BC at high doses are synergistic in patients with AIDS-related complex (ARC) and improve its symptoms, preventing the progress of the disease into the severe stage of AIDS; the same synergism helped also to increase the survival time in patients with

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severe AIDS.

CT Check Tags: Human

***Acquired Immunodeficiency Syndrome: TH, therapy**

***Hyperthermia, Induced: MT, methods**

***Neoplasms: TH, therapy**

Perfusion, Regional: MT, methods

L94 ANSWER 21 OF 108 MEDLINE

AN 96183933 MEDLINE

DN 96183933

TI Effect of whole-body hyperthermia on AIDS patients with Kaposi's sarcoma: a pilot study.

AU Steinhart C R; Ash S R; Gingrich C; Sapir D; Keeling G N; Yatvin M B

CS Mercy Special Immunology Services, Miami, Florida, USA.

SO JOURNAL OF ACQUIRED IMMUNE DEFICIENCY SYNDROMES AND HUMAN

RETROVIROLOGY, (1996 Mar 1) 11 (3) 271-81.

Journal code: B7J. ISSN: 1077-9450.

CY United States

DT (CLINICAL TRIAL)

Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199607

AB The safety and possible efficacy of extracorporeal whole-body hyperthermia (WBHT) were evaluated in the first FDA-approved feasibility study of WBHT in persons with AIDS. Six gay men, aged 20-50 years, CDC class C-3, underwent 1 h of WBHT at either 40 degrees C or 42 degrees C, employing a system that minimizes the physiological and biochemical changes that occur during WBHT. All subjects had Kaposi's sarcoma (KS), were free of opportunistic infections, and had significant elevations of plasma HIV RNA. During the treatment, there were no adverse side effects and all subjects tolerated WBHT without problems. KS lesions partially regressed immediately following WBHT in all subjects but returned to pretreatment status in five of six patients at 1 week. In subjects treated at 40 degrees C, CD4 counts decreased during the 8-week follow-up period; they remained unchanged, however, following 42 degrees C WBHT. Viral load remained unchanged following WBHT in subjects treated at 40 degrees C. Treatment at 42 degrees C resulted in an immediate reduction in HIV RNA that was not sustained at 1 week post-WBHT. We conclude that WBHT is safe in subjects with advanced HIV disease and that it may have a role in treating HIV infection. A larger controlled trial involving two treatments in less immunocompromised subjects is currently in progress to test this hypothesis.

CT Check Tags: Human; Male; Support, Non-U.S. Gov't

beta 2-Microglobulin: AN, analysis

Acquired Immunodeficiency Syndrome: BL, blood

Acquired Immunodeficiency Syndrome: CO, complications

***Acquired Immunodeficiency Syndrome: TH, therapy**

Adolescence

Adult

CD4 Lymphocyte Count

DNA, Viral: BL, blood

Follow-Up Studies

***Hyperthermia, Induced**

Hyperthermia, Induced: AE, adverse effects

HIV Core Protein p24: BL, blood

Middle Age

Pilot Projects

RNA, Viral: BL, blood

Sarcoma, Kaposi: CO, complications

***Sarcoma, Kaposi: TH, therapy**

CN 0 (beta 2-Microglobulin); 0 (DNA, Viral); 0 (HIV Core Protein p24);

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immunodeficiency virus type 1 and **Mycobacterium tuberculosis** infection in relation to tumor necrosis factor alpha prodn.)

L94 ANSWER 23 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 3

AN 96:328979 BIOSIS

DN 99051335

TI Antibiotics and increased temperature against *Borrelia burgdorferi* in vitro. X

AU Reisinger E; Wendelin I; Gasser R; Halwachs G; Wilders-Truschnig M; Krejs G

CS Dep. Med., Karl Franzens Univ., Auenbruggerplatz 15, A-8036 Graz, Austria

SO Scandinavian Journal of Infectious Diseases 28 (2). 1996. 155-157. ISSN: 0036-5548

LA English

PR Biological Abstracts Vol. 102 Iss. 003 Ref. 033508

AB In 1917, spirochaetal neurosyphilis was treated successfully with **malariotherapy** in combination with salvarsan or bismuth.

Malariotherapy for spirochaetal Lyme disease has been discussed, but the mechanism of an antispirochaetal effect remains unclear. We cultured *Borrelia burgdorferi* at different temperatures, alone and in combination with antibiotics. Our data demonstrate that growth of the strains PKo and ATCC 35210 (B31) was impaired at temperatures of 37 degree C and inhibited at 39 degree C and 40 degree C, respectively. Strain ATCC 35211, however, grew well up to 39 degree C but did not multiply at 40 degree C. A bactericidal effect was seen at 41 degree C for the strains B31 and PKo and at 42 degree C for all strains. The susceptibility of all strains to penicillin and ceftriaxone was increased up to 16-fold by an elevation of temperature from 36 degree C to 38 degree C. These in vitro data suggest that elevated body temperature may be beneficial during antimicrobial treatment of Lyme disease. This may be particularly important in tissues where high concentrations of antibiotics are difficult to achieve.

ST RESEARCH ARTICLE; BORRELIA BURGDOFFERI; PENICILLIN; ANTIBACTERIAL-DRUG; CEFTRIAXONE; ANTIBACTERIAL-DRUG; THERAPY

RN 1406-05-9 (PENICILLIN)

73384-59-5 (CEFTRIAXONE)

CC Biochemical Studies-General 10060

External Effects-Temperature as a Primary Variable *10614

Pathology, General and Miscellaneous-Therapy *12512

Physiology and Biochemistry of Bacteria *31000

In Vitro Studies, Cellular and Subcellular 32600

Medical and Clinical Microbiology-Bacteriology *36002

Chemotherapy-Antibacterial Agents *38504

BC Spirochaetaceae 06112

L94 ANSWER 24 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS

AN 96:106951 BIOSIS

DN 98679086

TI The katE gene, which encodes the catalase HPII of *Mycobacterium avium*.

AU Milano A; De Rossi E; Gusberti L; Heym B; Marone P; Riccardi G

CS Dipartimento Genetica Microbiologia, Univ. degli Studi Pavia, Via Abbiategrasso 207, 27100 Pavia, Italy

SO Molecular Microbiology 19 (1). 1996. 113-123. ISSN: 0950-382X

LA English

PR Biological Abstracts Vol. 101 Iss. 006 Ref. 079367

AB Disseminated *Mycobacterium avium*-*Mycobacterium intracellulare* disease is a prevalent opportunistic infection in patients with acquired immune deficiency syndrome (AIDS). These pathogens are generally resistant to isoniazid (INH), a powerful antituberculosis drug. It is now generally accepted that the INH susceptibility of

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Mycobacterium tuberculosis results from the transformation of the drug into a toxic derivative, as a result of the action of the enzyme catalase-peroxidase (HPI), encoded by the *katG* gene. It has been speculated that the presence of a second catalase (HPII) in some mycobacterial species, but lacking in **M. tuberculosis**, may impair the action of INH. In this report, the nucleotide sequence of the *M. avium katE* gene, encoding catalase HPII, is described. This enzyme shows strong similarity to *Escherichia coli* catalase HPII and eukaryotic catalases. All amino acids previously postulated as participating directly in catalysis by liver catalase and most of the amino acids binding the prosthetic group are conserved in *M. avium* catalase HPII. The enzyme is expressed in **E. coli** and is inhibited by 3-amino-1,2,4-triazole (AT). Furthermore, Southern blot hybridizations and polymerase chain reaction experiments demonstrate the distribution of *katE* gene in several mycobacterial species. To evaluate the potentially antagonistic effect of HPII catalase on INH susceptibility, the *katE* gene was transformed into **M. tuberculosis** H37Rv and the minimum inhibitory concentration (MIC) for INH was determined. Despite strong expression of the *katE* gene, no change in MIC was observed, thus ruling out a possible contribution of this enzyme to the natural resistance of *M. avium* to the drug. The availability of the gene probe, encoding the second mycobacterial catalase HPII, should open the way for the development of new drugs and diagnostic tests to combat drug-resistant pathogen strains.

ST RESEARCH ARTICLE; MYCOBACTERIUM AVIUM; MYCOBACTERIUM INTRACELLULARE; HUMAN; ACQUIRED IMMUNODEFICIENCY SYNDROME; OPPORTUNISTIC INFECTIONS; POLYMERASE CHAIN REACTION

RN 9001-05-2 (CATALASE)

CC Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Enzymes-Methods *10804
Enzymes-Physiological Studies *10808
Genetics of Bacteria and Viruses *31500
Immunology and Immunochemistry-Immunopathology, Tissue Immunology *34508
Medical and Clinical Microbiology-Bacteriology *36002
Medical and Clinical Microbiology-Virology *36006

BC Retroviridae 02623
Mycobacteriaceae 08881
Hominidae 86215

L94 ANSWER 25 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS

AN 96:399935 BIOSIS

DN 99122291

TI CD4 response in HIV+ patients treated with **malariotherapy**.

•AU Heimlich H J; Chen X P; Xiao B Q; Liu S G; Lu Y H; Spletzer E G; Yao J L

CS Heimlich Inst., Suite 410, 2368 Victory Pkwy., Cincinnati, OH 45206, USA

SO ELEVENTH INTERNATIONAL CONFERENCE ON AIDS. Eleventh International Conference on AIDS, Vol. Two. One world: One hope; Vancouver, British Columbia, Canada, July 7-12, 1996. viii+600p. Eleventh International Conference on AIDS: Vancouver, British Columbia, Canada 2 (0). 1996. 91.

DT Conference

LA English

PR Biological Abstracts/RRM Vol. 048 Iss. 009 Ref. 159868

ST MEETING ABSTRACT; MEETING POSTER; INTERLEUKIN; INTERFERON; HUMAN IMMUNODEFICIENCY VIRUS; ACQUIRED IMMUNODEFICIENCY SYNDROME; MORTALITY

CC General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520
Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous-Necrosis *12510

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Pathology, General and Miscellaneous-Therapy 12512
Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and
Reticuloendothelial System *15008
Endocrine System-General *17002
Immunology and Immunochemistry-Immunopathology, Tissue Immunology
*34508
Immunology, Parasitological *35000
Medical and Clinical Microbiology-Virology *36006
Parasitology-Medical *60504
BC Retroviridae 02623
Sporozoa 35400
Hominidae 86215

L94 ANSWER 26 OF 108 MEDLINE
AN 95367234 MEDLINE
DN 95367234
TI Severe ulcers from an unconventional therapy against AIDS [letter].
AU Santarossa S; Bernardi D; Tirelli U
SO AIDS, (1995 May) 9 (5) 536.
Journal code: AID. ISSN: 0269-9370.
CY United States
DT Letter
LA English
FS Priority Journals
EM 199511
CT Check Tags: Case Report; Human; Male; Support, Non-U.S. Gov't
***Acquired Immunodeficiency Syndrome: TH, therapy**
Adult
***Hyperthermia, Induced: AE, adverse effects**
***Skin Ulcer: ET, etiology**
Skin Ulcer: PA, pathology

L94 ANSWER 27 OF 108 MEDLINE
AN 96027809 MEDLINE
DN 96027809
TI Whole-body hyperthermia [letter; comment].
CM Comment on: J Acquir Immune Defic Syndr Hum Retrovirol 1995 Apr
1;8(4):321-9
AU Shecterle L M; St. Cyr J A
SO JOURNAL OF ACQUIRED IMMUNE DEFICIENCY SYNDROMES AND HUMAN
RETROVIROLOGY, (1995 Nov 1) 10 (3) 391.
Journal code: B7J. ISSN: 1077-9450.
CY United States
DT Commentary
Letter
LA English
FS Priority Journals
EM 199601
CT Check Tags: Human
***Acquired Immunodeficiency Syndrome: TH, therapy**
Clinical Trials, Phase I
***Hyperthermia, Induced: MT, methods**
***HIV Infections: TH, therapy**

L94 ANSWER 28 OF 108 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 4
AN 1995:967848 HCAPLUS
DN 124:75862
TI Thalidomide **inhibits** lipoarabinomannan-induced
upregulation of **human immunodeficiency** virus
expression
AU Peterson, Phillip K.; Gekker, Genya; Bornemann, Michel; Chatterjee,
Delphi; Chao, Chun C.
CS Dep. Med., Univ. Minnesota Med. Sch., Minneapolis, MN, USA
SO Antimicrob. Agents Chemother. (1995), 39(12), 2807-9
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CODEN: AMACCQ; ISSN: 0066-4804

DT Journal
 LA English
 CC 1-7 (Pharmacology)
 AB **Mycobacterium tuberculosis** accelerates the progression of **human** immunodeficiency virus type 1 (HIV-1) infection. The results of this study, which show that thalidomide **inhibits** the upregulation of **HIV-1** expression in U1 cells stimulated with mycobacterial lipoarabinomannans, support the rationale behind conducting controlled trials of this immunomodulatory agent with patients dually infected with HIV-1 and **M. tuberculosis**.
 ST thalidomide HIV1 **Mycobacterium tuberculosis** infection
 IT **Mycobacterium tuberculosis** (infection; thalidomide **inhibition** of lipoarabinomannan-induced upregulation of **HIV** expression in relation to dual **HIV-1** and **M. tuberculosis** infection **treatment**)
 IT Virus, animal (human immunodeficiency 1, infection; thalidomide **inhibition** of lipoarabinomannan-induced upregulation of **HIV** expression in relation to dual **HIV-1** and **M. tuberculosis** infection **treatment**)
 IT 50-35-1, Thalidomide
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses) (thalidomide **inhibition** of lipoarabinomannan-induced upregulation of **HIV** expression in relation to dual **HIV-1** and **M. tuberculosis** infection **treatment**)
 L94 ANSWER 29 OF 108 AIDSLINE
 AN 1996:4107 AIDSLINE
 DN MED-96155139
 TI A novel adjuvant for use with a blood-stage malaria vaccine.
 AU de Souza J B; Playfair J H
 CS University College London Medical School, Department of Immunology, UK.
 SO VACCINE, (1995). Vol. 13, No. 14, pp. 1316-9.
 Journal code: X60. ISSN: 0264-410X.
 CY ENGLAND: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 FS MED; Priority Journals
 LA English
 OS MEDLINE 96155139
 EM 199605
 AB An effective vaccine delivery system has been developed for vaccination against a blood-stage malaria infection in mice. Subcutaneous vaccination with a semi-purified asexual blood-stage malaria antigen combined with an adjuvant formulation containing squalane, Tween 80 and pluronic L121 (AF) protected mice infected with a lethal *P. yoelii* infection against death and greatly reduced the severity and duration of parasitaemia. The adjuvant and the route of immunization are both clinically acceptable, thereby making this an attractive delivery system for a human malaria vaccine. Protective immunity appeared to be associated with an enhancement of both Th1 and Th2 subset cytokines.
 CT Check Tags: Animal; Female; Male
 *Adjuvants, Immunologic: TU, therapeutic use
 Antibodies, Protozoan: BI, biosynthesis
 Antigens, Protozoan: IM, immunology
 CD8-Positive T-Lymphocytes: IM, immunology
 Injections, Subcutaneous

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Interferon Type II: ME, metabolism
 Interleukin-4: ME, metabolism
 Malaria: BL, blood
 Malaria: IM, immunology
 *Malaria: PC, prevention & control
***Malaria Vaccines: TU, therapeutic use**
 Mice
 Mice, Inbred BALB C
 Mice, Inbred C57BL
 *Plasmodium yoelii: IM, immunology
 Saponins: IM, immunology
 Saponins: TU, therapeutic use
 Spleen: ME, metabolism
 T-Lymphocytes, Cytotoxic: DE, drug effects
 T-Lymphocytes, Cytotoxic: IM, immunology
 Th1 Cells: IM, immunology
 Th2 Cells: IM, immunology
 RN 82115-62-6 (Interferon Type II)
 CN 0 (Adjuvants, Immunologic); 0 (Antibodies, Protozoan); 0 (Antigens, Protozoan); 0 (Interleukin-4); 0 (Malaria Vaccines); 0 (Saponins)

L94 ANSWER 30 OF 108 CANCERLIT
 AN 96113215 CANCERLIT
 DN 96113215
 TI Recent advances: antiinfectives.
 AU Briceland L L; Cleary J D; Fletcher C V; Healy D P; Peloquin C A
 CS Albany College of Pharmacy, NY, USA.
 SO ANNALS OF PHARMACOTHERAPY, (1995). Vol. 29, No. 10, pp. 1035-40.
 Journal code: BBX. ISSN: 1060-0280.
 DT Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 FS MEDL; L; Priority Journals
 LA English
 OS MEDLINE 96113215
 EM 199611
 AB OBJECTIVE: To update readers on the significant changes in infectious diseases pharmacotherapy. DATA SOURCES: An Index Medicus and Iowa Drug Information Service search (1993-1994) of English-language literature pertaining to the selected topic areas was performed. Additional information from abstracts presented at scientific meetings were identified by the authors. STUDY SELECTION AND DATA EXTRACTION: All identified studies were screened and those judged relevant to the update were evaluated. DATA SYNTHESIS: New or clinically significant data since 1992 that related to peptic ulcer disease, microbial resistance (e.g., Enterococcus spp., Streptococcus pneumoniae, Mycobacterium tuberculosis, Candida albicans), immunomodulators, and AIDS were evaluated and compared with previous data. CONCLUSIONS: There have been several exciting and significant changes in infectious diseases pharmacotherapy evident from this review. (49 Refs)

CT Check Tags: Comparative Study; Human
 Acquired Immunodeficiency Syndrome: DT, drug therapy
Adjuvants, Immunologic: TU, therapeutic use
 *Anti-Infective Agents: PD, pharmacology
Antiviral Agents: TU, therapeutic use
 Drug Resistance, Microbial
 Peptic Ulcer: DT, drug therapy
 Peptic Ulcer: MI, microbiology
 Streptococcus: DE, drug effects
 Tuberculosis: DT, drug therapy
Zidovudine: TU, therapeutic use
 RN 30516-87-1 (Zidovudine)
 CN 0 (Adjuvants, Immunologic); 0 (Anti-Infective Agents); 0 (Antiviral

08/846670

chimp
=> s human or ~~chip~~ or pig or monkey or mice

159865 HUMAN
93938 CHIP
14909 PIG
4182 MONKEY
31840 MICE
L1 267887 HUMAN OR *chimp* ~~CHIP~~ OR PIG OR MONKEY OR MICE

=> s hiv or cancer or lime or typhoid or norwalk or rotovirus

5221 HIV
21129 CANCER
24048 LIME
329 TYPHOID
2005 NORWALK
5 ROTOVIRUS
L2 50267 HIV OR CANCER OR LIME OR TYPHOID OR NORWALK OR ROTOVIRUS

=> s l1 and l2

L3 23038 L1 AND L2

=> s plasmodium or pallidum or smallpox or mycobacterium or ascaris or tapeworm or helicobacter or ulcer

798 PLASMODIUM
312 PALLIDUM
374 SMALLPOX
2867 MYCOBACTERIUM
628 ASCARIS
130 TAPEWORM
179 HELICOBACTER
4489 ULCER
L4 9105 PLASMODIUM OR PALLIDUM OR SMALLPOX OR MYCOBACTERIUM OR ASCARIS
RIS
OR TAPEWORM OR HELICOBACTER OR ULCER

=> s l3 and l4

L5 1796 L3 AND L4

=> s parasite

L6 2620 PARASITE

=> s l5 and l6

L7 228 L5 AND L6

=> d 17 1-228

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126. 5,403,934, Apr. 4, 1995, Heterocyclic compounds; John F. Batchelor, et al., 546/290, 296 [IMAGE AVAILABLE]

127. 5,403,484, Apr. 4, 1995, Viruses expressing chimeric binding proteins; Robert C. Ladner, et al., 435/235.1, 69.7, 172.3, 252.3, 320.1; 530/350; 536/23.4 [IMAGE AVAILABLE]

128. 5,389,368, Feb. 14, 1995, Avirulent microbes and uses therefor; Roy Gurtiss, III, 424/93.2, 93.4; 435/172.3, 320.1; 935/72, 73 [IMAGE AVAILABLE]

129. 5,387,744, Feb. 7, 1995, Avirulent microbes and uses therefor: Salmonella typhi; Roy Curtiss, III, et al., 424/235.1, 258.1; 435/172.3, 252.3, 252.33, 320.1, 879; 935/60, 62, 72 [IMAGE AVAILABLE]

130. 5,376,369, Dec. 27, 1994, Vaccine adjuvant; Anthony C. Allison, et al., 424/278.1, 279.1, 283.1; 436/543; 514/8, 885; 530/322, 806, 815 [IMAGE AVAILABLE]

131. 5,374,623, Dec. 20, 1994, Cysteine protease inhibitors effective for in vivo use; Mary P. Zimmerman, et al., 514/17; 530/330, 331, 332; 544/168; 560/10, 18, 37, 45 [IMAGE AVAILABLE]

132. 5,370,873, Dec. 6, 1994, Therapeutic compounds derived from the neem tree; Iroka J. Udeinya, 424/195.1; 514/896, 934 [IMAGE AVAILABLE]

133. 5,367,059, Nov. 22, 1994, Cys-Ser-Val-Thr-Cys-Gly specific tumor cell adhesion receptor; George P. Tuszynski, et al., 530/395, 350 [IMAGE AVAILABLE]

134. 5,356,927, Oct. 18, 1994, Methods of treating **plasmodium** and babesia parasitic infections; Theodore F. Taraschi, et al., 514/449, 895 ✓

[IMAGE AVAILABLE]

135. 5,356,797, Oct. 18, 1994, Membrane expression of heterologous genes; David W. Niesel, et al., 435/69.3, 69.1, 172.1, 172.3, 252.3, 320.1; 536/23.1, 23.7, 24.3 [IMAGE AVAILABLE]
136. 5,342,924, Aug. 30, 1994, Extracellular segments of **human** .epsilon. immunoglobulin anchoring peptides and antibodies specific therefor; Tse W. Chang, 530/387.9; 435/70.21; 530/388.1, 388.85, 389.1 [IMAGE AVAILABLE]
137. 5,338,842, Aug. 16, 1994, Yersinia INV nucleic acids; Ralph R. Isberg, et al., 536/23.7; 435/6, 69.1, 252.3, 252.33, 320.1; 536/24.32 [IMAGE AVAILABLE]
138. 5,334,379, Aug. 2, 1994, Cytokine and hormone carriers for conjugate vaccines; Subramonia Pillai, et al., 424/85.2, 85.1, 85.4, 197.11, 244.1, 250.1, 831; 530/351, 395, 404, 405, 406, 411 [IMAGE AVAILABLE]
139. 5,332,747, Jul. 26, 1994, Method for potentiating primary drugs in treating multidrug resistant parasitic disease cells; Knox Van Dyke, 514/280, 227.8, 281 [IMAGE AVAILABLE]
140. 5,332,567, Jul. 26, 1994, Detection and treatment of infections with immunoconjugates; M. David Goldenberg, 424/1.49, 1.53, 9.341, 136.1, 159.1, 164.1, 178.1 [IMAGE AVAILABLE]
141. 5,330,754, Jul. 19, 1994, Membrane-associated immunogens of mycobacteria; Archana Kapoor, et al., 424/190.1, 248.1; 435/69.3, 195; 514/2; 530/350; 536/23.7 [IMAGE AVAILABLE]
142. 5,328,930, Jul. 12, 1994, Treatment of microsporidial and acanthamoeba keratoconjunctivitis with topical fumagillin; Louis A. Wilson, 514/475, 912, 914 [IMAGE AVAILABLE]
143. 5,328,824, Jul. 12, 1994, Methods of using labeled nucleotides; David C. Ward, et al., 435/6, 7.1, 91.2; 536/22.1, 25.3, 25.32; 935/78 [IMAGE AVAILABLE]
144. 5,310,762, May 10, 1994, Medicaments; Victoria S. Latter, et al., 514/682 [IMAGE AVAILABLE]
145. 5,310,654, May 10, 1994, Method for determining virulence of Yersinia; Ralph R. Isberg, et al., 435/6; 536/23.7; 935/78 [IMAGE AVAILABLE]
146. 5,294,441, Mar. 15, 1994, Avirulent microbes and uses therefor: salmonella typhi; Roy Curtiss, III, 424/200.1, 235.1, 258.1; 435/172.3, 252.3, 252.33, 320.1, 879; 935/60, 62, 72 [IMAGE AVAILABLE]
147. 5,279,966, Jan. 18, 1994, Cloning, expression and uses of a novel secreted protein, F-spondin; Thomas M. Jessell, et al., 435/320.1, 69.1, 252.3; 530/395, 399; 536/23.5 [IMAGE AVAILABLE]
148. 5,278,173, Jan. 11, 1994, Method of inhibiting the activity of **human** immunodeficiency virus (HIV) in vivo; Michael H. Davis, 514/312, 885, 895 [IMAGE AVAILABLE]
149. 5,273,970, Dec. 28, 1993, Treatment of protozoal diseases; Nicholas McHardy, 514/157, 155, 158, 272 [IMAGE AVAILABLE]
150. 5,270,052, Dec. 14, 1993, Methods and compositions for treatment of infection by intracellular parasites; Jeffrey A. Gelfand, et al., 424/450; 436/829; 514/21 [IMAGE AVAILABLE]

151. 5,260,416, Nov. 9, 1993, Antigenic epitopes present on membrane-bound but not secreted IgE; Tse-wen Chang, 530/327; 424/131.1, 139.1, 140.1, 153.1, 805, 810; 530/387.2, 387.3, 388.73, 862, 868 [IMAGE AVAILABLE]
152. 5,254,671, Oct. 19, 1993, Extracellular segments of **human e** immunoglobulin anchoring peptides and antibodies specific therefor; Tse W. Chang, 530/324, 350, 386; 536/23.53 [IMAGE AVAILABLE]
153. 5,254,572, Oct. 19, 1993, Method and composition for supplementing vitamin B6 where the PN-PLP pathway is disturbed; Willem J. Serfontein, 514/345, 351 [IMAGE AVAILABLE]
154. 5,248,419, Sep. 28, 1993, Sewage sludge treatment with gas injection; Charles A. Long, Jr., et al., 210/218, 219; 261/89 [IMAGE AVAILABLE]
155. 5,246,930, Sep. 21, 1993, 9-substituted compounds of 3.alpha., 11.alpha.-epoxy-3,4,5,5a.alpha.,6,7,8,8a,9,11,11a-undecahydro-3.beta.,6.alpha.,9-trimethylfurano[3,4-j][1,2]benzodioxepin, processes for their preparation and their use as antiprotozoal and antiviral agents; Bindumadhavan Venugopalan, et al., 514/232.8, 253, 338, 348, 450; 544/148, 238, 378; 549/348 [IMAGE AVAILABLE]
156. 5,246,844, Sep. 21, 1993, Virulence associated proteins in *Borrelia burgdorferi* (BB); Steven J. Norris, et al., 435/172.3, 252.3, 252.33, 320.1; 536/23.7, 24.32, 24.33 [IMAGE AVAILABLE]
157. 5,246,596, Sep. 21, 1993, Method of treating waste to make it suitable for ultimate disposal; Philip N. Baldwin, Jr., et al., 210/750; 106/697; 210/764 [IMAGE AVAILABLE]
158. 5,239,066, Aug. 24, 1993, *Yersinia* ail nucleic acids; St. Geme, III: Joseph W., et al., 536/23.7; 435/6, 69.1, 252.3, 252.33, 320.1; 536/24.32; 935/11, 79 [IMAGE AVAILABLE]
159. 5,231,168, Jul. 27, 1993, Malaria antigen; Morten Dziegiel, et al., 530/350, 300 [IMAGE AVAILABLE]
160. 5,229,490, Jul. 20, 1993, Multiple antigen peptide system; James P. Tam, 530/324; 424/185.1, 186.1, 188.1, 189.1, 190.1, 191.1, 193.1, 196.11, 197.11; 530/323, 325, 326, 327, 328, 345, 403, 405, 409; 930/30, 210, 221 [IMAGE AVAILABLE]
161. 5,225,556, Jul. 6, 1993, Chemical probes for left-handed DNA and for A-DNA; chiral metal complexes as Z-specific antitumor agents and as double strand cleavers; Jacqueline K. Barton, 546/88; 204/157.71; 435/6, 52, 91.53, 810; 436/501; 536/23.1, 26.6; 546/10; 935/88 [IMAGE AVAILABLE]
162. 5,225,184, Jul. 6, 1993, Medicaments; Victoria S. Latter, et al., 424/45; 514/682 [IMAGE AVAILABLE]
163. 5,223,409, Jun. 29, 1993, Directed evolution of novel binding proteins; Robert C. Ladner, et al., 435/69.7, 5, 69.1, 172.3, 252.3, 320.1; 530/387.3, 387.5 [IMAGE AVAILABLE]
164. 5,217,898, Jun. 8, 1993, Expression of the *P. falciparum* transmission-blocking antigen in yeast; David C. Kaslow, et al., 435/254.2, 69.1, 69.3, 172.3, 235.1, 320.1; 530/350; 536/23.7; 935/10, 28, 37, 56, 65, 69 [IMAGE AVAILABLE]
165. 5,206,268, Apr. 27, 1993, Medicaments; Victoria S. Latter, et al., 514/548 [IMAGE AVAILABLE]

166. 5,198,347, Mar. 30, 1993, DNA encoding *Plasmodium vivax* and *Plasmodium knowlesi* Duffy receptor; Louis H. Miller, et al., 435/69.1, 252.3, 320.1; 530/350; 536/23.7 [IMAGE AVAILABLE]
167. 5,190,918, Mar. 2, 1993, Peptide fragments and analogs of thrombospondin and methods of use; Alan H. Deutch, et al., 514/15, 12, 13, 14; 530/324, 325, 326, 327, 328 [IMAGE AVAILABLE]
168. 5,185,146, Feb. 9, 1993, Recombinant MVA vaccinia virus; Werner Altenburger, 424/199.1, 232.1, 272.1; 435/69.1, 69.3, 172.1, 172.2, 172.3, 235.1, 236, 237, 239, 320.1; 935/12, 32, 57, 65 [IMAGE AVAILABLE]
169. 5,180,714, Jan. 19, 1993, Adenosine compounds for the treatment of diseases caused by parasitic protozoa; Janice R. Sufrin, et al., 514/46, 23, 45; 536/27.6 [IMAGE AVAILABLE]
170. 5,173,293, Dec. 22, 1992, Anti-T-cell antibodies as adjuvants; Sherree L. Friend, et al., 424/178.1, 154.1, 173.1, 193.1; 436/547, 548; 530/387.3, 388.22, 388.75, 389.6, 391.7, 403, 405, 406, 806, 807, 808, 809 [IMAGE AVAILABLE]
171. 5,169,862, Dec. 8, 1992, Analogs of viscosin and their uses; Terrence Burke, Jr., et al., 514/450; 530/321, 328; 549/351; 562/564, 577 [IMAGE AVAILABLE]
172. 5,157,024, Oct. 20, 1992, Method of enhancing the activity of phagocytes including macrophages, modulating the cellular or humoral immune response, and reducing the adverse effects of stress in warm blooded animals; Paul Gordon, 514/23, 25, 885, 889, 921; 536/17.4, 17.6, 17.9, 120 [IMAGE AVAILABLE]
173. 5,147,563, Sep. 15, 1992, Sewage sludge treatment with gas injection; Charles A. Long, Jr., et al., 210/758, 760, 764 [IMAGE AVAILABLE]
174. 5,112,869, May 12, 1992, Substituted 1-phenylnaphthalenes; Kyoichi A. Watanabe, et al., 514/641, 700, 717, 721, 732, 841, 842, 843, 883, 908; 564/270; 568/441, 632, 633, 734, 737, 808 [IMAGE AVAILABLE]
175. 5,112,749, May 12, 1992, Vaccines for the malaria circumsporozoite protein; Robert N. Brey, III, et al., 435/172.3, 69.1, 252.3, 320.1, 879; 530/350; 536/23.4, 23.7, 24.1; 935/12, 27, 41, 56, 65, 72 [IMAGE AVAILABLE]
176. 5,106,618, Apr. 21, 1992, Method of treating protozoal gastrointestinal disorders by administering hyperimmune milk product; Lee R. Beck, et al., 424/157.1, 163.1, 203.1, 535; 514/2, 8, 12, 21; 530/389.1, 389.5, 832 [IMAGE AVAILABLE]
177. 5,091,311, Feb. 25, 1992, The production of KSB-1939 macrolides using *STR eptomycetes hygroscopicus*; Hideki Katoh, et al., 435/119; 514/450 [IMAGE AVAILABLE]
178. 5,089,479, Feb. 18, 1992, Adhesion of *Mycoplasma pneumoniae* and *Mycoplasma hominus* to sulfatide; Howard C. Krivan, et al., 514/25; 435/101, 103, 176, 177, 182, 800, 870; 514/54, 59; 536/4.1, 112 [IMAGE AVAILABLE]
179. 5,087,453, Feb. 11, 1992, Method for the treatment of bacterial caused weight loss and/or hypoglycemia; Gideon Strassmann, 424/450, 85.1; 514/2; 530/399 [IMAGE AVAILABLE]
180. 5,041,385, Aug. 20, 1991, Vector expressing fusion proteins and particles; Alan J. Kingsman, et al., 435/320.1; 424/192.1, 210.1; 435/69.3, 69.7, 91.41, 170, 171, 172.1, 172.3, 235.1, 252.3, 254.21;

436/543; 536/23.4, 23.7; 935/9, 12, 22, 28, 47, 59, 60, 69 [IMAGE AVAILABLE]

181. 5,041,379, Aug. 20, 1991, Heliothis expression systems; Malcolm J. Fraser, et al., 435/235.1, 69.1, 70.1, 172.3, 320.1; 536/23.2, 23.6, 23.72; 935/3, 6, 9, 22, 33, 34, 47, 48, 59, 60, 61, 66, 70 [IMAGE AVAILABLE]

182. 5,030,200, Jul. 9, 1991, Method for eradicating infectious biological contaminants in body tissues; Millard M. Judy, et al., 604/5; 424/529 [IMAGE AVAILABLE]

183. 5,019,384, May 28, 1991, Immunomodulating compositions and their use; Malcolm L. Gefter, et al., 424/184.1, 185.1, 186.1, 190.1, 204.1, 234.1, 265.1, 272.1 [IMAGE AVAILABLE]

184. 5,008,373, Apr. 16, 1991, Fusion proteins and particles; Alan J. Kingsman, et al., 530/350; 435/69.7, 170, 171, 172.3, 233, 252.3, 254.2, 254.21, 320.1; 530/351, 412; 536/23.4; 935/10, 12, 22, 59, 66 [IMAGE AVAILABLE]

185. 4,981,874, Jan. 1, 1991, Medicaments; Victoria S. Latter, et al., 514/682 [IMAGE AVAILABLE]

186. 4,980,473, Dec. 25, 1990, Chemical probes for left-handed DNA and chiral metal complexes as Z-specific anti-tumor agents; Jacqueline K. Barton, 546/10; 987/5 [IMAGE AVAILABLE]

187. 4,963,354, Oct. 16, 1990, Use of tumor necrosis factor (TNF) as an adjuvant; H. Michael Shepard, et al., 424/85.1, 85.4; 514/2, 8, 12, 21, 885 [IMAGE AVAILABLE]

188. 4,946,849, Aug. 7, 1990, Method for the treatment of malaria; Michael T. Makler, 514/313 [IMAGE AVAILABLE]

189. 4,939,166, Jul. 3, 1990, Antibiotic KSB-1939 compounds as well as pesticidal agents containing same; Hideki Katoh, et al., 514/450; 549/264 [IMAGE AVAILABLE]

190. 4,939,088, Jul. 3, 1990, Sustained production of recombinant gamma interferon using an Epstein-Barr virus replicon; Janet M. Young, et al., 435/69.51; 424/85.5; 435/320.1, 364 [IMAGE AVAILABLE]

191. 4,925,831, May 15, 1990, Aminoalkyl naphthalenediols as host resistance enhancers against viral infection; Philippe L. Durette, 514/49, 42, 450, 459, 472, 552, 655 [IMAGE AVAILABLE]

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193. 4,906,564, Mar. 6, 1990, Antigenic determinants recognized by antibodies obtained using a pathogenic agent or a derivative thereof that presents a restricted set of antigens; Jeffery A. Lyon, et al., 435/7.22, 5, 29; 530/350, 388.6, 412, 413 [IMAGE AVAILABLE]

194. 4,902,431, Feb. 20, 1990, Method for treating wastewater sludge; John P. Nicholson, et al., 405/128; 71/13; 210/764, 916 [IMAGE AVAILABLE]

195. 4,900,722, Feb. 13, 1990, Methods and compositions for prophylactic and therapeutic treatment of infections; David L. Williams, et al., 514/54, 61; 536/117, 123.12, 124 [IMAGE AVAILABLE]

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655; 549/415, 424, 425, 472, 480, 492; 564/387 [IMAGE AVAILABLE]

197. 4,888,170, Dec. 19, 1989, Vaccines obtained from antigenic gene products of recombinant genes; Roy Curtiss, III, 424/200.1, 244.1, 258.1; 435/252.3, 252.8 [IMAGE AVAILABLE]

198. 4,886,743, Dec. 12, 1989, Diagnostic reagents based on unique sequences within the variable region of the T cell receptor and uses thereof; Leroy E. Hood, et al., 435/5, 6, 7.22, 7.23, 7.24, 29, 188, 974; 436/52, 63, 506, 508, 509, 536, 548, 813; 530/326, 387.9, 388.22, 388.75, 388.9, 389.1, 389.6, 389.8, 391.3; 536/24.3; 935/11, 12, 78, 104 [IMAGE AVAILABLE]

199. 4,878,891, Nov. 7, 1989, Method for eradicating infectious biological contaminants in body tissues; Millard M. Judy, et al., 604/5; 128/898; 424/529, 530, 531, 561 [IMAGE AVAILABLE]

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202. 4,835,177, May 30, 1989, Aminoalkyl naphthalenediols as host resistance enhancers against viral infection; Philippe L. Durette, 514/459, 472, 649, 650, 653 [IMAGE AVAILABLE]

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204. 4,793,927, Dec. 27, 1988, Method of treating sewage; Peter P. Meehan, et al., 405/128; 71/12, 901; 210/764 [IMAGE AVAILABLE]

205. 4,791,135, Dec. 13, 1988, Novel antimalarial dihydroartemisinin derivatives; Ai J. Lin, et al., 514/450; 549/348 [IMAGE AVAILABLE]

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207. 4,772,588, Sep. 20, 1988, Treatment of parasitic diseases with calf thymus extract; Giovanna Scioppacassi, 514/21; 424/580; 514/2, 8; 530/397, 399 [IMAGE AVAILABLE]

208. 4,772,466, Sep. 20, 1988, Vaccines comprising polyoxypropylene-polyoxyethylene block polymer based adjuvants; Anthony C. Allison, et al., 424/209.1, 207.1, 279.1, 280.1, 283.1 [IMAGE AVAILABLE]

209. 4,761,402, Aug. 2, 1988, Methods and compositions for prophylactic and therapeutic treatment of infections; David L. Williams, et al., 514/54, 25, 61; 536/117, 123.12, 124 [IMAGE AVAILABLE]

210. 4,746,612, May 24, 1988, Aotus interspecies hybridomas and monoclonal receptors produced thereby; Harold A. Stanley, et al., 530/388.6; 435/70.21, 172.2, 342; 530/350; 935/96, 100, 102, 103 [IMAGE AVAILABLE]

211. 4,744,984, May 17, 1988, Antiviral immunotherapeutic agent and preparation thereof; William L. Ragland, 424/282.1, 195.1, 283.1; 514/885, 937, 938 [IMAGE AVAILABLE]

212. 4,721,669, Jan. 26, 1988, Chemical probes for left-handed DNA and chiral metal complexes as Z-specific anti-tumor agents; Jacqueline K. Barton, 435/6; 204/157.72; 424/617; 536/25.4; 546/88; 935/78; 987/5 [IMAGE AVAILABLE]

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214. 4,714,606, Dec. 22, 1987, Method of staining and identifying cells and compositions thereof; Lawrence Kass, 435/40.51, 29, 34, 39; 534/611 [IMAGE AVAILABLE]

215. 4,711,955, Dec. 8, 1987, Modified nucleotides and methods of preparing and using same; David C. Ward, et al., 536/25.32, 25.6, 26.6 [IMAGE AVAILABLE]

216. 4,692,412, Sep. 8, 1987, Method of preparing an autogenous vaccine; Virginia W. Livingston, et al., 435/252.1; 424/234.1 [IMAGE AVAILABLE]

217. H 271, May 5, 1987, Treatment of malaria with esters of cephalotaxine; June M. Whaun, 514/214 [IMAGE AVAILABLE]

218. 4,574,058, Mar. 4, 1986, Antigen derivatives and processes for their preparation; Gerhard Baschang, et al., 536/17.2; 260/998.2; 530/322, 807; 536/17.3 [IMAGE AVAILABLE]

219. 4,532,122, Jul. 30, 1985, Anti-trypanosomal activity of platinum co-ordination compounds; Michael S. Wysor, et al., 424/649; 514/491, 922 [IMAGE AVAILABLE]

220. 4,510,144, Apr. 9, 1985, Methods of imparting immunomodulating activity with dihydrothiazolo purine derivatives; John W. Hadden, et al., 514/257, 267 [IMAGE AVAILABLE]

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222. 4,397,844, Aug. 9, 1983, Antigen derivatives and processes for their preparation; Gerhard Baschang, et al., 514/8; 530/806, 807; 536/53; 930/DIG.500 [IMAGE AVAILABLE]

223. 4,387,226, Jun. 7, 1983, Purine-dihydrothiazoles; John W. Hadden, et al., 544/247; 530/351; 544/251 [IMAGE AVAILABLE]

224. 4,375,542, Mar. 1, 1983, Kijanamicin antibiotics and derivatives thereof; Jay A. Waitz, et al., 536/7.1; 435/76; 514/27, 28, 29 [IMAGE AVAILABLE]

225. 4,235,995, Nov. 25, 1980, 3-Nitropyrazole derivatives; Reuben G. Jones, et al., 548/365.7; 546/275.4; 548/194, 364.7 [IMAGE AVAILABLE]

226. 4,145,554, Mar. 20, 1979, 3-Nitropyrazole derivatives; Reuben G. Jones, et al., 548/365.1, 364.7, 365.7, 371.7, 372.1 [IMAGE AVAILABLE]

227. 4,066,776, Jan. 3, 1978, Anti-bacterial compositions containing certain 3-nitropyrazoles; Reuben G. Jones, et al., 514/363, 339, 370, 407; 546/268.7, 275.4; 548/137, 197, 364.7, 365.7, 371.7, 372.5 [IMAGE AVAILABLE]

228. 3,958,025, May 18, 1976, Absciscic acid tablets and process; Virginia W-C Livingston, 514/557; 435/253.1 [IMAGE AVAILABLE]

3.2-fold (14 days after the tumor inoculation), whereas no change in the number of tumor-infiltrating lymphocytes was demonstrated in mice treated with Z-100 i.p. or i.v. as compared to controls. When BALB/c mice were inoculated s.c. with a mixture of Meth-A tumor cells (1×10^6) cells and lymphocytes (2×10^5) cells derived from Z-100-treated tumor tissues in a Winn's neutralization test, decreased growth of solid tumors was demonstrated as compared with that of control mice inoculated with tumor cells alone. However, no such inhibition of tumor growth was observed in mice inoculated with a mixture of the tumor cells and lymphocytes obtained from tumor tissues of control mice at the same effector to target cell ratio. (ABSTRACT TRUNCATED AT 250 WORDS)

CT Check Tags: Animal

***Adjuvants, Immunologic: TU, therapeutic use**

***Antineoplastic Agents: PD, pharmacology**

Drug Screening Assays, Antitumor

Injections

Interleukin-3: BI, biosynthesis

Lipids: IP, isolation & purification

***Lipids: PD, pharmacology**

Lymphocytes: DE, drug effects

Lymphocytes: IM, immunology

Mannans: IP, isolation & purification

***Mannans: PD, pharmacology**

Mice

Mice, Inbred BALB C

***Mycobacterium tuberculosis: CH, chemistry**

Sarcoma, Experimental: IM, immunology

***Sarcoma, Experimental: TH, therapy**

CN 0 (Adjuvants, Immunologic); 0 (Antineoplastic Agents); 0 (Interleukin-3); 0 (Lipids); 0 (Mannans); 0 (SSM)

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TI [Anti-infectious chemotherapy].

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DT Journal

FS 004 Microbiology

006 Internal Medicine

048 Gastroenterology

037 Drug Literature Index

LA French

SL French; English

AB Sarcoidosis and Crohn's disease may be due to a mycobacterium. PCR characterizes *Tropheryma whippelii*, the bacillary agent of Whipple's disease. Seven years or more after their introduction on the market, the fluoroquinolones are loosing activity against enterobacteriaceae, Salmonella, Campylobacter and even *E. coli*, due to the abuse of antibacterial agents by the alimentary industry. Intracellular kinetics allow prediction about the selective activity of macrolides and quinolones on intracellular pathogens. New data on *Helicobacter pylori*. Extended spectrum of the new macrolides to parasites and rickettsiae. How to treat *P. falciparum* malaria in pregnant women? Victories of qinghaosu derivatives and defeats of norfloxacin against *P. falciparum*. How to treat meningitis due to penicillin-cephalosporin-resistant pneumococci? Does chlorhexidin protect neonates against serious infections due to group B-streptococci? Severe Hib infections in the adult.

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Streptococcus sanguis or better Streptococcus sanguinis?

CT EMTAGS: **infection** (0310); **therapy** (0160); mammal (0738); **human** (0888); short survey (0002)

Medical Descriptors:

- *infection: DT, drug therapy
- *crohn disease: DT, drug therapy
- *malaria: DT, drug therapy

human

short survey

- *sarcoidosis: DT, drug therapy

Drug Descriptors:

- *quinoline derived antiinfective agent: DT, drug therapy
- *ciprofloxacin: DT, drug therapy
- *macrolide: DT, drug therapy
- *cephalosporin: DT, drug therapy
- *chlorhexidine: DT, drug therapy
- proguanil: DT, drug therapy
- norfloxacin: DT, drug therapy
- pyrimethamine: DT, drug therapy
- vancomycin: DT, drug therapy
- sulfadoxine: DT, drug therapy
- rifampicin: DT, drug therapy
- chloroquine: DT, drug therapy
- fosfomycin: DT, drug therapy
- clarithromycin: DT, drug therapy
- ceftriaxone: DT, drug therapy
- dirithromycin: DT, drug therapy
- roxithromycin: DT, drug therapy
- erythromycin: DT, drug therapy
- azithromycin: DT, drug therapy
- fleroxacin: DT, drug therapy
- mefloquine: DT, drug therapy
- pefloxacin: DT, drug therapy
- artemether: DT, drug therapy
- omeprazole: DT, drug therapy
- quinine: DT, drug therapy
- tetracycline: DT, drug therapy

RN 85721-33-1; 11111-12-9; 55-56-1; 3697-42-5; 500-92-5; 637-32-1; 70458-96-7; 58-14-0; 1404-90-6; 2447-57-6; 13292-46-1; 50-63-5; 54-05-7; 132-73-0; 3545-67-3; 23155-02-4; 81103-11-9; 73384-59-5; 74578-69-1; 62013-04-1; 80214-83-1; 114-07-8; 70536-18-4; 83905-01-5; 79660-72-3; 51773-92-3; 53230-10-7; 70458-92-3; 71963-77-4; 73590-58-6; 130-89-2; 130-95-0; 549-48-4; 7549-43-1; 60-54-8; 64-75-5

CN Quinodis

L94 ANSWER 51 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.

AN 93093053 EMBASE

TI The development and validation of a simple antigen detection ELISA for **Plasmodium falciparum** malaria.

AU Taylor D.W.; Voller A.

CS Department of Biology, Georgetown University, 37th and O Streets, Washington, DC 20057-1028, United States

SO TRANS. R. SOC. TROP. MED. HYG., (1993) 87/1 (29-31).
ISSN: 0035-9203 CODEN: TRSTAZ

CY United Kingdom

DT Journal

FS 004 Microbiology
017 Public Health, Social Medicine and Epidemiology

LA English

SL English

AB A double-antibody sandwich enzyme-linked immunosorbent assay (ELISA) is described for the detection of **Plasmodium falciparum** antigen. The test is based on an immunoglobulin

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(Ig) M capture monoclonal antibody on the solid phase and an IgG monoclonal antibody conjugated to peroxidase. The simple test takes about 2.5 h to complete and, because it uses whole blood with no prior **treatment**, it is possible to process batches of 50-100 samples simultaneously. The test is specific to **P. falciparum** and has a sensitivity close to that usually achieved with Giemsa-stained blood films. The reagents employed are stable at refrigerator temperatures for over 6 months, and as the test is compatible with human **immunodeficiency** virus and hepatitis B surface antigen ELISAs it could be suitable for blood transfusion screening.

CT EMTAGS: **immunological procedures** (0102);
invertebrate (0723); protozoon (0751); infection (0310);
 diagnosis (0140); methodology (0130); mammal (0738); **human**
 (0888); human tissue, cells or cell components (0111); priority
 journal (0007); article (0060); enzyme (0990)
 Medical Descriptors:
 *antigen detection
 ***plasmodium falciparum**
 *malaria: DI, diagnosis
 enzyme linked immunosorbent assay
 diagnostic accuracy
 diagnostic procedure
 screening
 methodology
human
 human tissue
 priority journal
 article
 Drug Descriptors:
 ***parasite antigen**
 immunoglobulin m: EC, endogenous compound
 monoclonal antibody
 peroxidase
 antibody conjugate
 RN 9007-85-6; 9003-99-0

L94 ANSWER 52 OF 108 AIDSLINE
 AN 1993:17184 AIDSLINE
 DN MED-93365402
 TI Quinolones in intracellular infections.
 AU Pech`ere J C
 CS Departement de Genetique et Microbiologie, Centre Medical
 Universitaire, Geneva, Switzerland.
 SO DRUGS, (1993). Vol. 45, Suppl. 3, pp. 29-36.
 Journal code: EC2. ISSN: 0012-6667.
 CY New Zealand
 DT Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, ACADEMIC)
 FS MED; Priority Journals
 LA English
 OS MEDLINE 93365402
 EM 199312
 AB Intracellular **parasites** are those which spend most of
 their lives within host cells. The fluoroquinolones demonstrate
 favourable intracellular pharmacokinetics for the treatment of
 intracellular infections; these agents diffuse and accumulate in the
 phagocytes, mainly in the cytosol, and do not associate with
 cellular organelles. The fluoroquinolones are generally active
 against *Salmonella* spp. in vitro, and have been used successfully in
 the treatment of typhoid fever, *Salmonella* bacteraemia in patients
 with AIDS, and chronic enteric carriage. Fluoroquinolone monotherapy
 has also been found satisfactory in the treatment of tularaemia and

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Mediterranean spotted fever. Quinolones, alone or in combination with other agents, have also shown promise in animal models of legionellosis and in limited clinical studies. Quinolones, particularly ciprofloxacin and ofloxacin, have notable antimycobacterial activity. Both agents have been used in combination with other antimycobacterial drugs in the treatment of infections caused by **Mycobacterium tuberculosis**, **M. avium-intracellulare** complex, rapidly growing mycobacteria and **M. leprae**, and deserve consideration as part of a multi-drug regimen in otherwise untreatable mycobacterial infections. Clinical data regarding fluoroquinolone monotherapy in brucellosis indicate unacceptable failure rates which preclude the use of these agents in this indication. The quinolones have some efficacy in genital chlamydial infections, but may have limitations in this indication also. In conclusion, as a result of the in vitro activity of the quinolones and their favourable pharmacokinetics, these agents are now an important part of the armamentarium against intracellular infections.

CT Check Tags: Animal; Human

Anti-Infective Agents, Fluoroquinolone: PK, pharmacokinetics

***Anti-Infective Agents, Fluoroquinolone: TU, therapeutic use**

Antibiotics: TU, therapeutic use

*Bacterial Infections: DT, drug therapy

Bacterial Infections: EP, epidemiology

Bacterial Infections: PP, physiopathology

Microbial Sensitivity Tests

CN 0 (Anti-Infective Agents, Fluoroquinolone); 0 (Antibiotics)

L94 ANSWER 53 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1993:94336 HCAPLUS

DN 118:94336

TI Treating infectious encephalitis with neuronal amino acid receptor-blocking agents

IN Bernton, Edward W.; Tortella, Frank C.

PA United States Dept. of the Army, USA

SO PCT Int. Appl., 34 pp.

CODEN: PIXXD2

PI WO 9221340 A1 921210

DS W: AT, AU, BB, BG, BR, CA, CH, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MG, MW, NL, NO, PL, RO, RU, SD, SE

RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR, IT, LU, MC, ML, MR, NL, SE, SN, TD, TG

AI WO 92-US4454 920527

PRAI US 91-710602 910605

DT Patent

LA English

IC ICM A61K031-44

ICS A61K031-215

CC 1-11 (Pharmacology)

AB Infectious and parainfectious encephalitis and encephalopathy from diverse causes, are treated with agents which block the neuronal excitatory amino acid receptor, specifically the N-methylaspartate binding receptor, or with other drugs which block amino acid excitotoxicity by inhibiting release of endogeneous excitatory amino acids. The drugs of choice are MK-801, dextromethorphan, carbetapentane, 7-chlorokynurenic acid, caramiphen, etc. The in-vitro degrading and lysis of fetal rat neurons by Mycoplasma fermentans was inhibited by pretreatment with MK-801.

ST encephalitis drug amino acid receptor antagonist

IT Acquired immune deficiency syndrome

Malaria

Reye's syndrome

Sepsis and Septicemia

(central nervous system dysfunction in, treatment of, with

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neuronal excitatory amino acid receptor-blocking agents)

IT **Escherichia coli**
 Haemophilus influenzae
 Mycoplasma fermentans
 Plasmodium **falciparum**
 Streptococcus
 Trypanosoma
 (encephalitis by, **treatment** of, with neuronal
 excitatory amino acid receptor-blocking agents)

IT Encephalitis
 (infectious and parainfectious, treatment of, by neuronal
 excitatory amino acid receptor-blocking agents)

IT Virus, animal
 (Epstein-Barr, encephalitis by, treatment of, with neuronal
 excitatory amino acid receptor-blocking agents)

IT Virus, animal
 (Japanese encephalitis, B, encephalitis by, treatment of, with
 neuronal excitatory amino acid receptor-blocking agents)

IT Virus, animal
 (St. Louis encephalitis, encephalitis by, treatment of, with
 neuronal excitatory amino acid receptor-blocking agents)

IT Virus, animal
 (Venezuelan equine encephalomyelitis, encephalitis by, treatment
 of, with neuronal excitatory amino acid receptor-blocking agents)

IT Virus, animal
 (arbo-, encephalitis by, treatment of, with neuronal excitatory
 amino acid receptor-blocking agents)

IT Virus, animal
 (cytomegalo-, encephalitis by, treatment of, with neuronal
 excitatory amino acid receptor-blocking agents)

IT Virus, animal
 (entero-, encephalitis by, treatment of, with neuronal excitatory
 amino acid receptor-blocking agents)

IT Virus, animal
 (herpes simplex 1, encephalitis by, treatment of, with neuronal
 excitatory amino acid receptor-blocking agents)

IT Neurotransmitter antagonists
 (methyl-D-aspartate, infectious and parainfectious encephalitis
 treatment by)

IT Virus, animal
 (rubella, encephalitis by, treatment of, with neuronal excitatory
 amino acid receptor-blocking agents)

IT Virus, animal
 (smallpox, encephalitis by, treatment of, with neuronal
 excitatory amino acid receptor-blocking agents)

IT Virus, animal
 (vaccinia, encephalitis by, treatment of, with neuronal
 excitatory amino acid receptor-blocking agents)

IT Virus, animal
 (varicella-zoster, encephalitis by, treatment of, with neuronal
 excitatory amino acid receptor-blocking agents)

IT Opioids
 RL: BIOL (Biological study)
 (.kappa-, antagonists of, neuronal protective, infectious and
 parainfectious encephalitis treatment by)

IT 77-22-5, Caramiphen 77-23-6 97-39-2 125-71-3, Dextromethorphan
 18000-24-3 77086-22-7, MK-801 115787-68-3, CI-972
 RL: BIOL (Biological study)
 (infectious and parainfectious encephalitis treatment by)

L94 ANSWER 54 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1993:11763 HCAPLUS

DN 118:11763

TI Liposomes coated with C-reactive proteins for treatment of infection

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by intracellular **parasites**

IN Gelfand, Jeffrey A.; Callahan, Michael V.; Yamada, Yoshinori
 PA New England Medical Center Hospitals, Inc., USA
 SO PCT Int. Appl., 20 pp.
 CODEN: PIXXD2
 PI WO 9218128 A1 921029
 DS W: CA, JP
 RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE
 AI WO 92-US3166 920416
 PRAI US 91-689709 910419
 DT Patent
 LA English
 IC ICM A61K031-47
 CC 63-6 (Pharmaceuticals)
 AB Liposomes contg. a drug directed against intercellular
parasites are coated with C-reactive proteins to efficiently
 target the drug to monocytes/macrophages. Liposomes manufd. with
 phosphatidylcholines and coated with C-reactive protein were
 equilibrated with hyperosmolar phosphate-buffered saline contg.
 amphotericin B (I) and sonicated to encapsulate I. Macrophage
 uptakes and anti-infective effects of the liposomes were studied.
 ST C reactive protein coating liposome target; amphotericin liposome C
 reactive protein coating; antiinfective liposome C reactive protein
 coating
 IT Chlamydia
 Leishmania tropica major
 Mycobacterium intracellulare
Mycobacterium tuberculosis
 (infection with, treatment of, with C-reactive protein-bound
 liposomes contg. drugs)
 IT **Parasite**
 (intracellular, infection with, treatment of, C-reactive
 protein-bound liposomes contg. drugs for)
 IT Bactericides, Disinfectants, and Antiseptics
 Fungicides and Fungistats
 Virucides and Virustats
 (liposomes contg., C-reactive protein-bound)
 IT Phosphatidylcholines, biological studies
 RL: BIOL (Biological study)
 (liposomes manuf. with, C-reactive protein coating in, for
 targeting monocyte/macrophages infected with intracellular
parasites)
 IT Proteins, specific or class
 RL: BIOL (Biological study)
 (C-reactive, anti-infective agent-contg. liposomes coating with,
 for targeting monocyte/macrophages)
 IT Virus, animal
 (human immunodeficiency 1, infection with,
treatment of, with C-reactive protein-bound liposomes
 contg. drugs)
 IT Pharmaceutical dosage forms
 (liposomes, C-reactive protein-bound, for targeting
 monocyte/macrophages infected with intracellular
parasites)
 IT 107-73-3, Phosphorylcholine
 RL: BIOL (Biological study)
 (liposomes manuf. with, C-reactive protein coating in, for
 targeting monocyte/macrophages infected with intracellular
parasites)
 IT 1397-89-3, Amphotericin B
 RL: BIOL (Biological study)
 (C-reactive protein-bound liposomes contg., for treatment of
 intracellular infections)

L94 ANSWER 55 OF 108 HCAPLUS COPYRIGHT 1998 ACS
 AN 1993:87601 HCAPLUS
 DN 118:87601
 TI Peptide epitopes of HIV gp120 conjugated to carriers as preventive vaccines for HIV
 IN Rubinstein, Arye; Bloom, Barry R.; Devash, Yair; Cryz, Stanley
 PA Schweiz. Serum- and Impfinstitut Bern, Switz.; Yeshiva University
 SO PCT Int. Appl., 64 pp.
 CODEN: PIXXD2
 PI WO 9217590 A1 921015
 DS W: AU, CA, JP
 RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE
 AI WO 92-EP735 920402
 PRAI US 91-681624 910402
 US 92-837781 920214
 DT Patent
 LA English
 IC ICM C12N015-49
 ICS A61K039-21; G01N033-569
 CC 63-3 (Pharmaceuticals)
 AB The title gp120 epitope-carrier conjugates for use as HIV vaccines are claimed. After vaccination with the conjugates, antibody-contg. fluid is extd. from individuals and assessed in an antigen-limited ELISA which contains a thimerosal-contg. diluent and selects for high affinity/avidity neutralizing and/or protective HIV-specific antibodies. The conjugates which have induced the prodn. of such antibodies are useful in the **treatment** and transmission prevention of **HIV**. Conjugates of purified protein deriv. of tuberculin from **Mycobacterium tuberculosis** with gp120 epitopes were prepd. and administered to 5 humans. After a 3rd immunization, one volunteer had a high titer of high affinity/high avidity HIV-specific antibodies. Upon exposure to the HIV epitope, the lymphocytes of this individual responded in vitro by proliferation and secretion of interleukin-2.
 ST HIV vaccine gp120 epitope carrier conjugate
 IT Mycobacterium BCG
 (conjugates, with HIV gp120 epitopes, prepn. and use of, as HIV vaccine)
 IT Hemocyanins
 RL: PREP (Preparation)
 (keyhole limpet, conjugates, with HIV gp120 epitopes, prepn. and use as HIV vaccine of)
 IT Vaccines
 (to HIV, gp120 epitope-immunogenic carrier conjugates as, prepn. of)
 IT Tuberculins
 RL: PREP (Preparation)
 (PPD (purified protein derivs.), conjugates, with HIV gp120 epitopes, prepn. and use as HIV vaccine of)
 IT Immunostimulants
 (adjuvants, Freund's, adjuvant, for HIV gp120 epitope-immunogenic carrier conjugate, HIV vaccine in relation to)
 IT Immunostimulants
 (adjuvants, ISCOMs, adjuvant, for HIV gp120 epitope-immunogenic carrier conjugate, HIV vaccine in relation to)
 IT Immunostimulants
 (adjuvants, Ribi, adjuvant, for HIV gp120 epitope-immunogenic carrier conjugate, HIV vaccine in relation to)
 IT Polyesters, biological studies
 RL: BIOL (Biological study)
 (dilactone-based, HIV gp120 epitope-immunogenic carrier conjugate microencapsulation with, HIV vaccine in relation to)
 IT Toxoids
 RL: PREP (Preparation)

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(diphtheria, conjugates, with HIV gp120 epitopes, prepn. and use as HIV vaccine of)

IT Toxins
 RL: PREP (Preparation)
 (exo-, A, of Pseudomonas aeruginosa, conjugates, with HIV gp120 epitopes, prepn. and use as HIV vaccine of)

IT Sialoglycoproteins
 RL: PREP (Preparation)
 (gp120env, epitopes of, of HIV, conjugates with immunogenic carriers of, prepn. and use as HIV vaccines of)

IT Antigens
 RL: PREP (Preparation)
 (hepatitis B core, conjugates, with HIV gp120 epitopes, prepn. and use as HIV vaccine of)

IT Virus, animal
 (human immunodeficiency, vaccines for, gp120 epitope-immunogenic carrier conjugates as, prepn. of)

IT Glycophospholipids
 RL: BIOL (Biological study)
 (lipid A, monophosphates, adjuvant, for HIV gp120 epitope-immunogenic carrier conjugate, HIV vaccine in relation to)

IT Encapsulation
 (micro-, of HIV gp120 epitope-immunogenic carrier conjugate, HIV vaccine in relation to)

IT Toxoids
 RL: PREP (Preparation)
 (tetanus, conjugates, with HIV gp120 epitopes, prepn. and use as HIV vaccine of)

IT Organelle
 (virosome, adjuvant, for HIV gp120 epitope-immunogenic carrier conjugate, HIV vaccine in relation to)

IT 7784-30-7 21645-51-2, Aluminum hydroxide (Al(OH)₃), biological studies
 RL: BIOL (Biological study)
 (adjuvant for HIV gp120 epitope-immunogenic carrier conjugates, HIV vaccine in relation to)

IT 1344-28-1, Alumina, biological studies
 RL: BIOL (Biological study)
 (adjuvant, for HIV gp120 epitope-immunogenic carrier conjugate, HIV vaccine in relation to)

IT 54-64-8, Thimerosal
 RL: BIOL (Biological study)
 (in assay for anti-HIV antibodies, selection of vaccine in relation to)

IT 128554-25-6DP, conjugate with immunogenic carrier 128554-26-7DP, conjugate with immunogenic carrier 128554-28-9DP, conjugate with immunogenic carrier 128554-29-0DP, conjugate with immunogenic carrier 128554-31-4DP, conjugate with immunogenic carrier 128554-34-7DP, conjugate with immunogenic carrier 128554-35-8DP, conjugate with immunogenic carrier 128554-38-1DP, conjugate with immunogenic carrier 130036-94-1DP, conjugate with immunogenic carrier 131474-06-1DP, conjugate with immunogenic carrier 145785-52-0DP, conjugate with immunogenic carrier 145785-53-1DP, conjugate with immunogenic carrier 145785-54-2DP, conjugate with immunogenic carrier
 RL: SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and use of, as HIV vaccine)

L94 ANSWER 56 OF 108 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
 AN 92-041352 [05] WPIDS
 CR 92-041346 [05]
 DNC C92-018097
 TI Pure transfer factor with activity greater than 5,000 units per
 KATHLEEN FULLER BT/LIBRARY 308-4290

AU-214 - used to treat viral, bacterial and protozoal infections
e.g. HIV, herpes and candida.

DC B04 C06 D16

IN KIRKPATRICK, C H; ROZZO, S J; KIRKPATRICK, C H

PA (NAJE-N) NAT JEWISH CENT IMMUNOLOGY & RESPIRATORY; (NAJE-N) NAT
JEWISH CENT IMM; (NAJE-N) NAT JEWISH CENT IMM

CYC 33

PI WO 9200093 A 920109 (9205)*
RW: AT BE CH DE DK ES FR GB IT LU NL OA SE
W: AT AU BB BG BR CA CH DE DK ES FI GB HU JP KP KR LK LU MC MG
MW NL NO RO SD SE SU

AU 9181957 A 920227 (9218)

JP 05508847 W 931209 (9403) 21 pp C07K015-06

AU 657915 B 950330 (9521) C07K015-06

EP 537280 B1 970917 (9742) EN 40 pp A61K038-00
R: AT BE CH DE DK ES FR GB GR IT LI LU NL SE

DE 69127694 E 971023 (9748) A61K038-00

ADT JP 05508847 W JP 91-512313 910702, WO 91-US4779 910702; AU 657915 B
AU 91-81957 910702; EP 537280 B1 EP 91-913547 910702, WO 91-US4779
910702; DE 69127694 E DE 91-627694 910702, EP 91-913547 910702, WO
91-US4779 910702

FDT JP 05508847 W Based on WO 9200093; AU 657915 B Previous Publ. AU
9181957, Based on WO 9200093; EP 537280 B1 Based on WO 9200093; DE
69127694 E Based on EP 537280, Based on WO 9200093

PRAI US 91-718571 910626; US 90-547500 900702

REP 6.Jnl.Ref ; EP 101200; EP 143445; US 3991132; US 4468372; US 4616079

IC A61K037-02; C07K003-00
ICM A61K038-00; C07K015-06
ICS A61K037-02; C07K001-00; C07K003-00; C07K003-18

AB WO 9200093 A UPAB: 950609
A pure transfer factor (TF) with a specific activity of at least
5000 units per absorbance unit at 214nm is claimed.
Also claimed are (A) a pure TF with a mol.wt. of 4500-5500
daltons as determined by aminoacid analysis, which migrates as a
single peak on reverse phase, which has a specific activity of at
least 5000 units per absorbance unit at 214nm; (B) a method of
producing pure TF; (C) a method of treating a **human** of
animal with an infection caused by a microorganism comprising
administering a pure TF specific for the microorganism with a
specific activity of at least 5,000 units per absorbance unit at
214nm; and (D) a method of preventing an infection in a
human or animal by a microorganism comprising administering
a pure TF specific for the microorganism with a specific activity of
at least 5,000 units per absorbance unit at 214nm.
USE/ADVANTAGE - The pure TF is effective in transferring cell
mediated immunity to **humans** or animals. The TFs activate
the cell mediated immune system and act very rapidly to prevent or
treat infection caused by viruses, e.g. Herpes simplex or HIV, fungi
e.g. Candida albicans, bacteria e.g. **Mycobacterium**
tuberculosis, **parasites**, e.g. coccidia or
protozoa. @ (69pp Dwg.No.0/0

FS CPI

FA AB

MC CPI: B04-B04A1; B12-A01; B12-A02C; B12-A04; B12-A06; B12-B04;
C04-B04A1; C12-A01; C12-A02C; C12-A04; C12-A06; C12-B04;
D05-H13

L94 ANSWER 57 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 7

AN 92:441619 BIOSIS

DN BR43:74619

TI THE HISTORY OF **MALARIOOTHERAPY** FOR NEUROSYPHILIS MODERN
PARALLELS.

AU AUSTIN S C; STOLLEY P D; LASKY T

CS DEP. EPIDEMIOL. AND PREVENTIVE MED., UNIV. MD. SCH. MED., 600 REDWOOD
KATHLEEN FULLER BT/LIBRARY 308-4290

$$\begin{array}{c} \text{W} \quad \quad \text{W} \\ || \quad \quad || \\ \text{R}-\text{W}-\text{P}-\text{W}-\text{P}-\text{W} \\ | \quad \quad | \\ \text{WR1} \quad \text{WR2} \end{array} \left[\begin{array}{ccc} & \text{X} & \text{Y} \\ & \diagdown \quad \diagup & \\ \text{A} & & \text{B} \\ | & \text{---} & | \\ \text{Z} & & \text{D} \\ | & & | \\ \text{C} & & \end{array} \right] \quad \text{I}$$

- AB 5'-Diphosphohexose nucleosides I (A, B, C = H, halo, azido; D = H, halo, azido, OH; A and B or C and D can be replaced by a double bond; R = aldohexose, aldohexosamine, N-acetyl aldohexosamine; R1, R2 = H, C1-10 alkyl; W = O, S; X = O, S, CH2; Y = purine, pyrimidine base, Z = C, S, O; if Z = S, O, A and C are not present) are prepd. that have enhanced pharmaceutical or biol. activity or increased intracellular absorption compared to the corresponding parent nucleoside as a function of the 5'-diphosphohexose moiety. Many of these compds. have antiviral, including anti-AIDS virus, activity. Others have antibacterial activity. In one embodiment, a method is described to **treat human immunodeficiency virus (HIV) infection** and opportunistic infections concomitantly. 3'-Azido-2',3'-dideoxyuridine-5'-diphospho-N-acetylglucosamine (prepn. described) had a median effective concn. (EC50) of 0.02-0.41 .mu.M against HIV-1 in vitro. The 50% inhibitory concn. (IC50) of this compd. against normal, uninfected **human** peripheral blood mononuclear cells was >100 .mu.M. The compd. also inhibited
- ST phosphohexose nucleoside antiviral antibacterial; AIDS virus phosphahexose nucleoside
- IT Nucleosides, biological studies
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(biol. activity of, enhancement of, by derivatizing with diphosphohexose)
- IT Anti-infective agents
(diphosphohexose nucleosides)
- IT Pharmaceutical dosage forms
(diphosphohexose nucleosides in, as antimicrobials)
- IT Macrophage
(nucleoside conversion to antimicrobial diphosphohexose deriv. in)
- IT Cryptococcus neoformans
Histoplasma capsulatum
Legionella
Mycobacterium intracellulare
Mycobacterium tuberculosis
Mycoplasma
Pneumocystis carinii pneumoniae
Salmonella
Shigella
Toxoplasma
(opportunistic infection with, **treatment** of, with diphosphohexose nucleosides)
- IT Molecular structure-biological activity relationship
(Staphylococcus aureus-inhibiting, of azidodideoxyuridine derivs.)
- IT Virus, animal
(cytomegalo-, opportunistic infection with, treatment of, with diphosphohexose nucleosides)
- IT Virus, animal
(**human immunodeficiency**, infection with, **treatment** of, with diphosphohexose nucleosides)
- IT Virus, animal
(**human immunodeficiency 1, inhibition** of, with azidodideoxyuridinediphosphohexoses, in **human** peripheral blood mononuclear cells)
- IT Pharmaceutical dosage forms
(liposomes, diphosphohexose nucleosides in, as antimicrobials)
- IT Bactericides, Disinfectants, and Antiseptics
Fungicides and Fungistats

(medical, diphosphohexose nucleosides)

IT Leukocyte
(mononuclear, azidodideoxyuridine metab. in)

IT 3056-17-5D, 3'-Deoxy-2',3'-didehydrothymidine, diphosphohexose
derivs. 4097-22-7D, 2',3'-Dideoxyadenosine, diphosphohexose
derivs. 7481-88-1D, diphosphohexose derivs. 7481-89-2D,
2',3'-Dideoxycytidine, diphosphohexose derivs. 21679-14-1D,
9-.beta.-D-Arabinofuranosyl-2-fluoroadenine, diphosphohexose derivs.
25526-93-6D, 3'-Fluoro-3'-deoxythymidine, diphosphohexose derivs.
28446-21-1D, nucleoside derivs. 30516-87-1D, 3'-Azido-3'-
deoxythymidine, diphosphohexose derivs. 41107-56-6D,
diphosphohexose derivs. 69123-90-6D, diphosphohexose derivs.
69304-47-8D, diphosphohexose derivs. 69655-05-6D,
2',3'-Dideoxyinosine, diphosphohexose derivs. 77181-69-2D,
diphosphohexose derivs. 83546-42-3D, diphosphohexose derivs.
84472-85-5D, 3'-Azido-2',3'-dideoxyuridine, diphosphohexose derivs.
85326-07-4D, diphosphohexose derivs. 85326-07-4D, halo,
diphosphohexose derivs. 87190-79-2D, diphosphohexose derivs.
105380-83-4D, diphosphohexose derivs. 115249-95-1D,
diphosphohexose derivs. 134680-32-3D, diphosphohexose derivs.
136465-73-1D, diphosphohexose derivs.
RL: BIOL (Biological study)
(antimicrobials)

IT 9024-82-2, Inorganic pyrophosphatase 9026-22-6,
UDPG-pyrophosphorylase
RL: BIOL (Biological study)
(in prepn. of antiviral azidodideoxyuridinediphosphoglucose)

IT 132278-28-5P 132278-29-6P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and anti-**human** immunodeficiency virus activity
and toxicity of)

IT 5983-03-9P 14270-73-6P 84472-84-4P 84472-85-5P,
3'-Azido-2',3'-dideoxyuridine 117783-53-6P 136491-33-3P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction of, in prepn. of antimicrobial
diphosphohexose deriv.)

IT 136465-75-3P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of, in prepn. of antimicrobial diphosphohexose deriv.)

IT 59-56-3, Glucose-1-phosphate 119388-79-3
RL: RCT (Reactant)
(reaction of, in enzymic prepn. of antiviral deriv.)

IT 951-78-0, 2'-Deoxyuridine 73577-59-0
RL: RCT (Reactant)
(reaction of, in prepn. of antimicrobial diphosphohexose deriv.)

IT 136465-79-7
RL: RCT (Reactant)
(reaction of, in prepn. of antimicrobial diphosphohexose
nucleoside deriv.)

IT 136465-76-4
RL: PRP (Properties)
(toxicity of, in cultured **human** peripheral blood
mononuclear cells)

L94 ANSWER 59 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1991:243590 HCAPLUS
DN 114:243590
TI Detection and treatment of infections with immunoconjugates and
sterile injectable preparations for targeting infections
IN Goldenberg, Milton David
PA Immunomedics, Inc., USA
SO Eur. Pat. Appl., 20 pp.
CODEN: EPXXDW
PI EP 417927 A1 910320

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DS R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE
 AI EP 90-309319 900824
 PRAI US 89-399566 890824
 DT Patent
 LA English
 IC ICM A61K049-02
 ICS A61K047-48; A61K049-00; A61K043-00
 CC 8-9 (Radiation Biochemistry)
 Section cross-reference(s): 15, 63
 AB Diagnostic or therapeutic agent conjugates with (a) an antibody or antibody fragment which binds to an epitope on a pathogen or an antigen derived therefrom, or (b) an immunoreactive composite of chem.-linked antibodies or fragments binding to such epitopes are used in the detection or treatment of infections. A sterile, injectable prepn. for such use is also provided. **Mice** were hyperimmunized with glycoprotein gp160 of the AIDS virus and monoclonal antibodies MAb-160s1 and MAb-160s2 plus others were prepd. by the hybridoma method. The Fab' fragment of MAb-160s1 was prepd. and conjugated with 99mTc or with 131I and Fab' fragment of MAb-160s2. The conjugates were used in SPECT imaging and AIDS therapy, resp.
 ST infection immunoconjugate diagnosis therapy; antibody AIDS virus immunoconjugate; pathogen antibody immunoconjugate; imaging AIDS virus antibody conjugate
 IT Therapeutics
 (agents for, conjugates with antibodies to pathogens, for targeting infection foci)
 IT Anti-infective agents
 (anti-pathogen antibody conjugates with therapeutic agents as)
 IT Antigens
 RL: BIOL (Biological study)
 (antibodies to, of pathogens, conjugates with diagnostic or therapeutic agents, for targeting infection foci)
 IT *Acholeplasma laidlawii*
Babesia bovis
Brucella abortus
Echinococcus granulosus
Elmeria tenella
Escherichia coli
Legionella pneumophila
Leishmania tropica
Mesocystoides corti
Mycobacterium leprae
Mycobacterium tuberculosis
Mycoplasma arginini
Mycoplasma arthritidis
Mycoplasma hyorhinis
Mycoplasma orale
Mycoplasma pneumoniae
Mycoplasma salivarium
Mycoplasma
Neisseria gonorrhoeae
Neisseria meningitidis
Onchocerca volvulus
Plasmodium falciparum
Plasmodium vivax
 Protozoa
Pseudomonas aeruginosa
Schistosoma japonicum
Schistosoma mansoni
Streptococcus agalactiae
Streptococcus pneumoniae
Streptococcus pyogenes
Taenia hydatigena

Taenia ovis
Taenia saginata
Theileria parva
Toxoplasma gondii
Treponema pallidum
Trichinella spiralis
Trypanosoma brucei
Trypanosoma cruzi
Trypanosoma rangeli
Trypanosoma rhodesiense
 (antibody to, conjugates with diagnostic or therapeutic agents,
 for targeting infection foci)
IT Cytotoxic agents
 (conjugates with anti-pathogen antibodies, for targeting
 infection foci)
IT Lymphokines and Cytokines
RL: BIOL (Biological study)
 (hematopoietic toxicity prevention by, in formulation contg.
 therapeutic agent-antibody conjugate)
IT Virus, animal
 (human serum parvo-like, antibody to, conjugates with
 diagnostic or therapeutic agents, for targeting infection foci)
IT Anthelmintics
Antimalarials
Protozoacides
Virucides and Virustats
 (infection-targeting antibody-therapeutic agent conjugates as)
IT Infection
 (targeting of, with antibody conjugates with diagnostic or
 therapeutic agents)
IT Antiserums
 (to pathogen, conjugates with diagnostic or therapeutic agents,
 for targeting infection foci)
IT Antibodies
RL: BIOL (Biological study)
 (to pathogen, conjugates with diagnostic or therapeutic agents,
 for targeting infection foci)
IT Hematopoietic precursor cell
 (toxicity to, by therapeutic agent-antibody conjugate, cytokine
 protection against)
IT Malaria
 (treatment of, with anti-malaria antibody-pyrimethamine
 conjugate)
IT Leprosy
 (treatment of, with iodine-131-radioiodinated antibody
 conjugates)
IT Virus, animal
 (DNA-contg., antibody to, conjugates with diagnostic or
 therapeutic agents, for targeting infection foci)
IT Virus, animal
 (Epstein-Barr, antibody to, conjugates with diagnostic or
 therapeutic agents, for targeting infection foci)
IT Spirochaetales
 (Lyme disease, antibody to, conjugates with diagnostic or
 therapeutic agents, for targeting infection foci)
IT Imaging
 (NMR, agents, for magnetic resonance image enhancement,
 conjugates with anti-pathogen antibodies, for targeting infection
 foci)
IT Virus, animal
 (RNA-contg., antibody to, conjugates with diagnostic or
 therapeutic agents, for targeting infection foci)
IT Virus, animal
 (SV40, antibody to, conjugates with diagnostic or therapeutic

agents, for targeting infection foci)

IT Virus, animal
(Sendai, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Virus, animal
(Sindbis, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT **Immunodeficiency**
(acquired **immune deficiency** syndrome, **treatment** of, with iodine-131-anti-glycoprotein gp160 monoclonal antibody fragment conjugate)

IT Virus, animal
(adeno-, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Diagnosis
(agents, conjugates with antibodies to pathogens, for targeting infection foci)

IT Virus, animal
(bluetongue, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Radioelements, compounds
RL: BIOL (Biological study)
(conjugates, with antibodies to pathogens, for targeting infection foci)

IT Virus, animal
(cytomegalo-, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Virus, animal
(dengue, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Virus, animal
(feline leukemia, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Glycoproteins, specific or class
RL: SPN (Synthetic preparation); PREP (Preparation)
(gp160env, monoclonal antibodies to, prepn. of, for prep. diagnostic imaging and therapeutic conjugates)

IT Virus, animal
(hepatitis B, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Virus, animal
(herpes, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Virus, animal
(**human** T-cell leukemia, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Virus, animal
(**human** immunodeficiency, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Virus, animal
(**human** immunodeficiency 1, glycoprotein gp160 of, monoclonal antibodies to, prepn. of, for prep. diagnostic imaging and therapeutic conjugates)

IT Virus, animal
(**human** wart, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Scintigraphy
(immuno-, of AIDS virus-pos. patient, technetium-99m-labeled anti-glycoprotein gp160 antibody Fab' fragment in)

IT Virus, animal
(influenza, antibody to, conjugates with diagnostic or therapeutic agents, for targeting infection foci)

IT Pharmaceutical dosage forms
(injections, of antibody conjugates with diagnostic or

therapeutic agents, for targeting infection foci)

IT Virus, animal
(lymphocytic choriomeningitis, antibody to, conjugates with
diagnostic or therapeutic agents, for targeting infection foci)

IT Virus, animal
(measles, antibody to, conjugates with diagnostic or therapeutic
agents, for targeting infection foci)

IT Bactericides, Disinfectants, and Antiseptics
(medical, infection-targeting antibody-therapeutic agent
conjugates as)

IT Antibodies
RL: BIOL (Biological study)
(monoclonal, to pathogen, conjugates with diagnostic or
therapeutic agents, for targeting infection foci)

IT Virus, animal
(mumps, antibody to, conjugates with diagnostic or therapeutic
agents, for targeting infection foci)

IT Virus, animal
(murine leukemia, antibody to, conjugates with diagnostic or
therapeutic agents, for targeting infection foci)

IT Virus, animal
(murine mammary tumor, antibody to, conjugates with diagnostic or
therapeutic agents, for targeting infection foci)

IT Microorganism
(pathogenic, antibody to, conjugates with diagnostic or
therapeutic agents, for targeting infection foci)

IT Virus, animal
(polio-, antibody to, conjugates with diagnostic or therapeutic
agents, for targeting infection foci)

IT Virus, animal
(rabies, antibody to, conjugates with diagnostic or therapeutic
agents, for targeting infection foci)

IT Virus, animal
(reo-, antibody to, conjugates with diagnostic or therapeutic
agents, for targeting infection foci)

IT Virus, animal
(respiratory syncytial, antibody to, conjugates with diagnostic
or therapeutic agents, for targeting infection foci)

IT Virus, animal
(rubella, antibody to, conjugates with diagnostic or therapeutic
agents, for targeting infection foci)

IT Tomography
(single-photon-emission, computerized, of AIDS virus-pos.
patient, technetium-99m-labeled anti-glycoprotein gp160 antibody
Fab' fragment in)

IT Toxins
RL: BIOL (Biological study)
(tetanus, antibody to, conjugates with diagnostic or therapeutic
agents, for targeting infection foci)

IT Haemophilus influenzae
(type b, antibody to, conjugates with diagnostic or therapeutic
agents, for targeting infection foci)

IT Virus, animal
(varicella-zoster, antibody to, conjugates with diagnostic or
therapeutic agents, for targeting infection foci)

IT Virus, animal
(vesicular stomatitis, antibody to, conjugates with diagnostic or
therapeutic agents, for targeting infection foci)

IT 23288-61-1D, monoclonal antibody fragment conjugates
RL: BIOL (Biological study)
(AIDS virus infection foci imaging with)

IT 10043-66-0D, Iodine-131, bivalent monoclonal antibody fragment
conjugates
RL: BIOL (Biological study)

(AIDS virus infection foci treatment with)
IT 7440-42-8D, Boron, adducts, anti-pathogen antibody conjugates
RL: BIOL (Biological study)
(infection diagnosis or treatment with)
IT 58-14-0D, Pyrimethamine, monoclonal antibody fragments conjugates
RL: BIOL (Biological study)
(malaria therapy with)

L94 ANSWER 60 OF 108 MEDLINE
AN 92015631 MEDLINE
DN 92015631
TI From the Centers for Disease Control. Self-induced malaria X
associated with **malariotherapy** for Lyme disease--Texas.
AU Anonymous
SO JAMA, (1991 Oct 23-30) 266 (16) 2199.
Journal code: KFR. ISSN: 0098-7484.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199201
CT Check Tags: Animal; Case Report; Human; Male
***Hyperthermia, Induced: AE, adverse effects**
*Lyme Disease: TH, therapy
*Malaria: ET, etiology
*Plasmodium vivax
Texas

L94 ANSWER 61 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
AN 91338896 EMBASE
TI Self-induced malaria associated with **malariotherapy** for X
Lyme disease - Texas.
AU Rawlings J.; Perdue J.N.; Perrotta D.; Simpson D.
CS Division of Parasitic Diseases, Malaria Branch, National Center for
Infectious Diseases, CDC, Atlanta, GA, United States
SO J. AM. MED. ASSOC., (1991) 266/16 (2199).
ISSN: 0098-7484 CODEN: JAMAAP
CY United States
DT Journal
FS 004 Microbiology
037 Drug Literature Index
LA English
CT EMTAGS: infection (0310); etiology (0135); therapy (0160); North
America (0405); invertebrate (0723); protozoon (0751); mammal
(0738); human (0888); male (0041); case report (0151); priority
journal (0007); note (0063)
Medical Descriptors:
*lyme arthritis: ET, etiology
*lyme arthritis: DT, drug therapy
*malaria: ET, etiology
*malaria: DT, drug therapy
*arthralgia: ET, etiology
united states
plasmodium vivax
human
male
case report
priority journal
note
Drug Descriptors:
*chloroquine: DT, drug therapy

RN 50-63-5; 54-05-7; 132-73-0; 3545-67-3

L94 ANSWER 62 OF 108 HCAPLUS COPYRIGHT 1998 ACS
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AN 1992:34006 HCAPLUS
 DN 116:34006
 TI Enhancement of monocyte antimycobacterial activity by
 diethyldithiocarbamate (DTC)
 AU Huebner, L.; Ernst, M.; Von Laer, D.; Schwander, S.; Flad, H. D.
 CS Dep. Immunol. Cell Biol., Forschungsinst. Borstel, Borstel, D-2061,
 Germany
 SO Int. J. Immunopharmacol. (1991), 13(8), 1067-72
 CODEN: IJIMDS; ISSN: 0192-0561
 DT Journal
 LA English
 CC 1-5 (Pharmacology)
 AB Diethyldithiocarbamate (DTC) has been recently reported to
 significantly reduce the incidence of opportunistic infections in
 HIV-infected patients. The present study addresses the question
 whether DTC is capable of stimulating antimycobacterial activity of
 mononuclear phagocytes. The authors found that peripheral blood
 mononuclear cells (PBMC) of healthy subjects preincubated in vitro
 with 100-1000 ng/mL of DTC and thereafter infected with
Mycobacterium tuberculosis H37Rv or M.
 avium-intracellular complex exhibited an enhanced antimycobacterial
 activity compared with control-incubated cells as assessed by the
 detn. of mycobacterial colony-forming units. In subsequent expts.
 monocytes from healthy volunteers injected with 5 mg/kg body wt. of
 DTC weretested ex vivo for antimycobacterial activity at various
 periods of time after injection. Injection of DTC resulted in a
 significant enhancement of antimycobacterial activity which was most
 evident 24 h after DTC injection. The authors conclude that DTC
 stimulates the antimicrobial function of mononuclear phagocytes both
 in vitro and in vivo. These results may explain the favorable clin.
 course obsd. in HIV-infected patients **treated**
 with DTC and may serve as a basis for treatment with DTC in patients
 with drug-resistant atypical mycobacteriosis.
 ST diethyldithiocarbamate monocyte Mycobacterium infection AIDS
 IT Monocyte
 (antimycobacterial activity of, diethyldithiocarbamate
 enhancement of, of normal and HIV-infected **humans**)
 IT Bactericides, Disinfectants, and Antiseptics
 (ciethyldithiocarbamate, monocyte antimycobacterial activity
 enhancement by, of normal and HIV-infected **humans**)
 IT Acquired immune deficiency syndrome
 (diethyldithiocarbamate enhancement of antimycobacterial activity
 of monocytes from **humans** with)
 IT Mycobacterium avium
Mycobacterium tuberculosis
 (infection with, of monocyte, growth inhibition in,
 diethyldithiocarbamate enhancement of, of normal and HIV-infected
humans)
 IT 147-84-2, biological studies
 RL: BIOL (Biological study)
 (monocyte antimycobacterial activity enhancement by, of normal
 and HIV-infected **humans**)

L94 ANSWER 63 OF 108 MEDLINE
 AN 91375391 MEDLINE
 DN 91375391
 TI Update: self-induced malaria associated with **malariotherapy** *
 for Lyme disease--Texas.
 AU Anonymous
 SO MMWR. MORBIDITY AND MORTALITY WEEKLY REPORT, (1991 Oct 4) 40 (39)
 665-6.
 Journal code: NE8. ISSN: 0149-2195.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)

LA English
 FS Priority Journals
 EM 199112
 AB In December 1990, the Texas Department of Health (TDH) was contacted by a man who had recently moved from the northeastern United States and who was considering **malariotherapy** for Lyme disease (LD). He described a 2-year history of unsuccessful treatment with multiple antibiotics for arthralgias and palpitations, which had been diagnosed as LD.

CT Check Tags: Animal; Case Report; Human; Male
***Hyperthermia, Induced: AE, adverse effects**
 *Lyme Disease: TH, therapy
 *Malaria: ET, etiology
 *Plasmodium vivax
 Texas

L94 ANSWER 64 OF 108 HCAPLUS COPYRIGHT 1998 ACS
 AN 1991:677344 HCAPLUS
 DN 115:277344
 TI Surface expression of malarial antigens in E. coli and S. typhimurium: induction of serum antibody response upon oral vaccination of mice
 AU Schorr, Joachim; Knapp, Bernhard; Hundt, Erika; Kuepper, Hans; Amann, Egon
 CS Res. Lab., Behringwerke A.-G., Marburg, D-3550, Fed. Rep. Ger.
 SO Vaccines 91: Mod. Approaches New Vaccines Incl. Prev. AIDS, [Annu. Meet. Mod. Approaches New Vaccines], 8th (1991), Meeting Date 1990, 387-92. Editor(s): Chanock, Robert M. Publisher: Cold Spring Harbor Lab., Plainview, N. Y.
 CODEN: 57HGAV
 DT Conference
 LA English
 CC 15-2 (Immunochemistry)
 AB The Escherichia coli OmpA protein can serve as a carrier for the expression of foreign antigens at the surface of gram-neg. bacteria. OmpA vectors were used to express immunogenic segments of the protective Plasmodium **falciparum** blood-stage antigens SERP and HRPII in E. coli and Salmonella typhimurium. Upon induction, the malaria-specific sequences of 189 (HRPII) and 451 (SERP) amino acids, fused into the E. coli OmpA protein, were expressed. Immunofluorescence studies, immunogold-staining expts., and trypsin **treatment** of live **E. coli** cells expressing the HRPII-OmpA and SERP-OmpA fusion proteins demonstrate the surface exposition of these malarial antigens. Oral vaccination of mice with a Salmonella vaccine strain expressing the malarial antigens at its surface resulted in the induction of specific serum IgG antibodies. Thus, the OmpA surface expression system in combination with Salmonella vaccine strains can be used to deliver efficiently large antigens to the mucosal immune system.

ST antigen malaria expression Salmonella Escherichia
 IT Vaccines
 (antibody response to malaria antigen expression in
 microorganisms in relation to)
 IT Malaria
 (antigens in, expression of, in microorganism, antibody response
 in relation to)
 IT Plasmodium **falciparum**
 (antigens of, expression of, in microorganism, malaria vaccine
 and antibody response in relation to)
 IT Escherichia coli
 Salmonella typhimurium
 (malaria antigen expression in, antibody response in relation to)
 IT Antibodies
 RL: PRP (Properties)

(malaria antigen induction of, antigen expression in microorganisms in relation to)

IT Antigens
 RL: BIOL (Biological study)
 (of malaria, expression of, in microorganism, antibody response in relation to)

L94 ANSWER 65 OF 108 MEDLINE DUPLICATE 9
 AN 91080259 MEDLINE
 DN 91080259
 TI From the Centers for Disease Control. Imported malaria associated with **malariotherapy** of Lyme disease--New Jersey.
 AU Anonymous
 SO JAMA, (1991 Jan 16) 265 (3) 317-8.
 Journal code: KFR. ISSN: 0098-7484.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
 EM 199104
 CT Check Tags: Animal; Human
 ***Hyperthermia, Induced: AE, adverse effects**
 *Lyme Disease: TH, therapy
 Malaria: EP, epidemiology
 *Malaria: ET, etiology
 New Jersey: EP, epidemiology
 *Plasmodium vivax

L94 ANSWER 66 OF 108 HCAPLUS COPYRIGHT 1998 ACS
 AN 1991:179675 HCAPLUS
 DN 114:179675
 TI Functional expression of the dihydrofolate reductase and thymidylate synthetase activities of the human malaria parasite Plasmodium **falciparum** in Escherichia coli
 AU Hall, Stephen J.; Sims, Paul F. G.; Hyde, John E.
 CS Inst. Sci. Technol., Univ. Manchester, Manchester, M60 1QD, UK
 SO Mol. Biochem. Parasitol. (1991), 45(2), 317-30
 CODEN: MBIPDP; ISSN: 0166-6851
 DT Journal
 LA English
 CC 3-4 (Biochemical Genetics)
 Section cross-reference(s): 10
 AB A recombinant system was developed that directs the functional expression from Escherichia coli of both dihydrofolate reductase-thymidylate synthetase (DHFR-TS) and the isolated DHFR domain from Plasmodium **falciparum**. Both products are **inhibitory** to a no. of **E. coli** cell lines to the extent that cell growth ceases immediately upon induction. This dramatic inhibition is not seen in strain AB1899, in which amts. of plasmodial protein of up to 100 times the basal **E. coli** TS level can be accumulated. However, as well as the full-length DHFR-TS mol., smaller proteins carrying an intact TS substrate-binding site are produced. These represent ca. 60-75% of the total plasmodial protein expressed and are obsd. in every **E. coli** strain examd. They are not derived by degrdn. of the parent DHFR-TS mol., but can be correlated with the sizes of proteins expected to be produced if erroneous initiation of translation were occurring at 3 internal methionine residues.
 ST Plasmodium dihydrofolate reductase gene cloning Escherichia
 IT Escherichia coli
 (cloning and expression in, of dihydrofolate reductase and thymidylate synthetase genes of Plasmodium **falciparum**)
 IT Plasmodium **falciparum**
 (dihydrofolate reductase-thymidylate synthetase gene of, cloning
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and expression of, in *Escherichia coli*)
 IT Gene and Genetic element, microbial
 RL: BIOL (Biological study)
 (for dihydrofolate reductase and thymidylate synthetase, of
Plasmodium falciparum, cloning and expression in
Escherichia coli of)

IT Molecular cloning
 (of dihydrofolate reductase and thymidylate synthetase genes, of
Plasmodium falciparum, in *Escherichia coli*)

IT 9002-03-3, Dihydrofolate reductase 9031-61-2, Thymidylate
 synthetase
 RL: PRP (Properties)
 (gene for, of *Plasmodium falciparum*, cloning and
 expression in *Escherichia coli* of)

L94 ANSWER 67 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 91113940 EMBASE
 TI Epidemiologic notes and reports: Imported malaria associated with
malariotherapy of Lyme disease - New Jersey.
 AU Mertz K.
 CS New Jersey State Department of Health, Division of Parasitic
 Diseases, Center for Infectious Diseases, Trenton, NJ, United States
 SO ARCH. DERMATOL., (1991) 127/2 (161).
 ISSN: 0003-987X CODEN: ARDEAC
 CY United States
 DT Journal
 FS 004 Microbiology
 013 Dermatology and Venereology
 017 Public Health, Social Medicine and Epidemiology
 037 Drug Literature Index
 LA English
 CT EMTAGS: epidemiology (0400); infection (0310); therapy (0160);
 mammal (0738); human (0888); priority journal (0007); note (0063)
 Medical Descriptors:
 *epidemiology
 *malaria: DT, drug therapy
 *lyme arthritis
 human
 priority journal
 note
 Drug Descriptors:
 *chloroquine: DT, drug therapy

RN 50-63-5; 54-05-7; 132-73-0; 3545-67-3

L94 ANSWER 68 OF 108 CANCERLIT
 AN 92121627 CANCERLIT
 DN 92121627
 TI EFFECTS OF ACETYL-L-CARNITINE ORAL ADMINISTRATION ON LYMPHOCYTE
 ANTIBACTERIAL ACTIVITY AND TNF-ALPHA LEVELS IN PATIENTS WITH ACTIVE
 PULMONARY TUBERCULOSIS. A RANDOMIZED DOUBLE BLIND VERSUS PLACEBO
 STUDY.
 AU Jirillo E; Altamura M; Munno I; Pellegrino N M; Sabato R; Di Fabio
 S; De Simone C
 CS Cattedra di Immunologia, Universita di Bari, Italy.
 SO IMMUNOPHARMACOLOGY AND IMMUNOTOXICOLOGY, (1991). Vol. 13, No. 1-2,
 pp. 135-46.
 Journal code: IAI. ISSN: 0892-3973.
 DT (CLINICAL TRIAL)
 Journal; Article; (JOURNAL ARTICLE)
 (RANDOMIZED CONTROLLED TRIAL)
 FS MEDL; L; Priority Journals
 LA English
 OS MEDLINE 92121627
 EM 199203


AB Acetyl-L-carnitine (ALC), a drug for the treatment of ageing-related neuroendocrine dysfunctions, was orally administered--2 gm/day for 30 days--to 10 patients with active pulmonary tuberculosis (TBC). Lymphocyte-mediated antibacterial activity and serum levels of tumor necrosis factor (TNF)-alpha were evaluated before and after treatment, comparing the values with those of 10 TBC patients receiving placebo. Results show that by day 30, antibacterial activity remained unmodified or increased in ALC-treated subjects, while decreased in the placebo group. No influence of ALC on TNF-alpha levels was detectable. These data suggest that the host's immune responses to **M. tuberculosis** infection can be selectively modulated by drugs acting on the neuroendocrine axis.


CT Check Tags: Female; Human; Male
Acetylcarnitine: AD, administration & dosage
***Acetylcarnitine: TU, therapeutic use**
Adjuvants, Immunologic: AD, administration & dosage
Adjuvants, Immunologic: TU, therapeutic use
Administration, Oral
Adult
Aged
Blood Bactericidal Activity: DE, drug effects
Double-Blind Method
Lymphocytes: DE, drug effects
Lymphocytes: IM, immunology
Middle Age
***Tuberculosis, Pulmonary: DT, drug therapy**
Tuberculosis, Pulmonary: IM, immunology
Tumor Necrosis Factor: ME, metabolism
RN 14992-62-2 (Acetylcarnitine)
CN 0 (Adjuvants, Immunologic); 0 (Tumor Necrosis Factor)

L94 ANSWER 69 OF 108 MEDLINE
AN 90220775 MEDLINE
DN 90220775
TI Should we try **malariotherapy** for Lyme disease? [letter].
AU Heimlich H J
SO NEW ENGLAND JOURNAL OF MEDICINE, (1990 Apr 26) 322 (17) 1234-5.
Journal code: NOW. ISSN: 0028-4793.
CY United States
DT Letter
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199007
CT Check Tags: Human
***Hyperthermia, Induced**
***Lyme Disease: TH, therapy**
Malaria: IM, immunology
Neurosyphilis: TH, therapy

L94 ANSWER 70 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1991:178376 HCAPLUS
DN 114:178376
TI Synergistic rifampicin-based drug compositions for treatment of mycobacterial diseases
IN Freerksen, Enno Prof Dr Dr
PA Saarsstickstoff-Fatol G.m.b.H., Fed. Rep. Ger.
SO Ger. Offen., 6 pp.
CODEN: GWXXBX
PI DE 3911263 A1 901011
AI DE 89-3911263 890407
DT Patent
LA German
IC ICM A61K031-63

ICS A61K031-505; A61K031-495; A61K031-44
 ICI A61K031-63, A61K031-505, A61K031-495, A61K031-44
 CC 1-5 (Pharmacology)
 AB Cotrifazide (rifampicin-sulfamethoxazole-trimethoprim-isoniazid
 mixt.) and emdetin (rifampicin-sulfamethoxazole-trimethoprim-
 protionamide mixt.) are synergistic drugs for the treatment of
 mycobacterial diseases, opportunistic infections in AIDS, leprosy,
 malaria and hospitalism. Repeated oral administration of
 cotrifazide decreased the serum activity of Mycobacterium marinum,
M. tuberculosis and M. avium, in **humans**.
 ST bactericide mycobacteria cotrifazide emdetin; AIDS opportunistic
 infection cotrifazide emdetin; leprosy drug cotrifazide emdetin;
 malaria drug cotrifazide emdetin
 IT Leprosy
 Malaria
 (treatment of, with cotrifazide and emdetin)
 IT **Immunodeficiency**
 (acquired **immune deficiency** syndrome,
treatment of mycobacterial infections in, with
 cotrifazide and emdetin)
 IT Bactericides, Disinfectants, and Antiseptics
 (medical, cotrifazide and emdetin, for treatment of mycobacterial
 diseases)
 IT 133468-04-9 133468-05-0
 RL: BIOL (Biological study)
 (mycobacterial diseases treatment by)

L94 ANSWER 71 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 90138206 EMBASE
 TI Should we try **malariotherapy** for Lyme disease?. 
 AU Heimlich H.J.
 CS Heimlich Institute, Cincinnati, OH 45208, United States
 SO NEW ENGL. J. MED., (1990) 322/17 (1234-1235).
 ISSN: 0028-4793 CODEN: NEJMAG
 CY United States
 DT Journal
 FS 004 Microbiology
 008 Neurology and Neurosurgery
 LA English
 CC 037.11.04.00.00. Drug Literature Index/ANTIINFECTIVE
 AGENTS/Antiprotozoal drugs
 CT EMTAGS: infection (0310); therapy (0160); nervous system (0910);
 human (0888); bacterium (0762); letter (0008); priority journal
 (0007)
 Medical Descriptors:
 *syphilis: DT, drug therapy
 *lyme arthritis: DT, drug therapy
 *malaria: DT, drug therapy
 nervous system
 tumor necrosis factor
 interleukin 1
 Drug Descriptors:
 *antimalarial agent: DT, drug therapy

L94 ANSWER 72 OF 108 MEDLINE
 AN 91056807 MEDLINE 
 DN 91056807
 TI Imported malaria associated with **malariotherapy** of Lyme
 disease--New Jersey.
 AU Anonymous
 SO MMWR. MORBIDITY AND MORTALITY WEEKLY REPORT, (1990 Dec 7) 39 (48)
 873-5.
 Journal code: NE8. ISSN: 0149-2195.
 CY United States

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199103
CT Check Tags: Animal; Human
*Hyperthermia, Induced: AE, adverse effects
*Lyme Disease: TH, therapy
Malaria: EP, epidemiology
*Malaria: ET, etiology
New Jersey: EP, epidemiology
*Plasmodium vivax

L94 ANSWER 73 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS

AN 90:215658 BIOSIS

DN BA89:112948

TI BACTERICIDAL ACTIVITY IN-VITRO OF VARIOUS RIFAMYCINS AGAINST
MYCOBACTERIUM-AVIUM AND MYCOBACTERIUM-TUBERCULOSIS

AU HEIFETS L B; LINDHOLM-LEVY P J; FLORY M A

CS NATIONAL JEWISH CENTER IMMUNOL. RESPIRATORY MED., 1400 JACKSON ST.,
DENVER, COLO. 80206.

SO AM REV RESPIR DIS 141 (3). 1990. 626-630. CODEN: ARDSBL ISSN:
0003-0805

LA English

AB Minimal **inhibitory** and bactericidal concentrations (MICs
and MBCs) of rifampin (RMP), rifabutin (RBT), rifapentine (RPT),
CGP-7040, and P-DEA, were determined for 50 M. avium strains in 7H12
liquid medium radiometrically under various pH conditions. Half were
isolated from patients with AIDS and the other half from patients
without AIDS but with pulmonary disease. The MICs and MBCs were also
determined in 7H12 broth for **M. tuberculosis**
strains. The MIC results obtained with **M.**
tuberculosis strains, and the serum peak levels in
humans, were used as standards for interpretation of the MICs
and MBCs of the rifamycins for M. avium. The bactericidal activity of
all rifamycins for M. avium was substantially lower than for
M. tuberculosis. The majority of M. avium strains
was within the "susceptible" category, e.g., comparable to
susceptible **M. tuberculosis** strains, when tested
with CGP-7040 and RPT, and all of them were "moderately susceptible"
when tested with P-DEA. On the basis of in vitro bacteriostatic and
bactericidal activity, it seems that three agents, RPT, P-DEA, and
CGP-7040 have more potential than do RMP and RBT against M. avium
disease.

ST **HUMAN** RIFAMPIN RIFABUTIN RIFAPENTINE CGP-7040 P-DEA
ANTIBACTERIAL-DRUG ACQUIRED **IMMUNE DEFICIENCY**
SYNDROME PULMONARY DISEASE MINIMUM **INHIBITORY** CONCENTRATION
MINIMUM BACTERICIDAL CONCENTRATION

RN 13292-46-1 (RIFAMPIN)
13553-79-2 (RIFAMYCINS)
61379-65-5 (RIFAPENTINE)
72559-06-9 (RIFABUTIN)
122188-44-7 (CGP-7040)

CC Biochemical Studies-General 10060
Pathology, General and Miscellaneous-Therapy *12512
Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and
Reticuloendothelial Pathologies 15006
Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and
Reticuloendothelial System 15008
Respiratory System-General; Methods 16001
Respiratory System-Pathology *16006
Pharmacology-Clinical Pharmacology *22005
Virology-Animal Host Viruses 33506
Immunology and Immunochemistry-Immunopathology, Tissue Immunology

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*34508

Medical and Clinical Microbiology-Bacteriology *36002

Medical and Clinical Microbiology-Virology *36006

Chemotherapy-Antibacterial Agents *38504

BC Retroviridae-Lentivirinae 02242

Mycobacteriaceae 05822

Hominidae 86215

L94 ANSWER 74 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.

AN 90204660 EMBASE

TI The clinical and **parasitological** presentation of**Plasmodium falciparum** malaria in Uganda isunaffected by **HIV-1** infection.

AU Muller O.; Moser R.

CS German Red Cross Society, Baerwaldstrasse 55, 1000 Berlin 61,

Germany, Federal Republic of

SO TRANS. R. SOC. TROP. MED. HYG., (1990) 84/3 (336-338).

ISSN: 0035-9203 CODEN: TRSTAZ

CY United Kingdom

DT Journal

FS 004 Microbiology

047 Virology

LA English

AB The relation between **Plasmodium falciparum**malaria and symptomatic human **immunodeficiency** virus 1 (**HIV-1**) infection was investigated in paediatric and adult

patients in Kampala, Uganda, from 1987 to 1989. Both infections

contributed largely to hospital morbidity. Of 1527 clinically

suspicious in-patients, 61% were positive for **HIV-1**infection. 52% of patients with positive **HIV-1** serology

fulfilled the World Health Organization clinical case definition for

acquired **immune deficiency** syndrome (AIDS) inAfrica. No association could be found between **HIV-1**infection and malaria either in paediatrics or in adults. **P**. **falciparum parasitaemia** was present in 18% of

all patients and no differences in prevalence of malaria infection

or in **parasite** density could be demonstrated between**HIV-1** positive and **HIV-1** negative patients. The

comparison of clinical symptoms showed typical differences in

AIDS-related morbidity but no difference in malaria-specific

morbidity. Also, the response to malaria **treatment** was thesame in **HIV-1** positive and **HIV-1** negativepatients. **P. falciparum** malaria does not appear

to act as an opportunistic agent in AIDS patients in Uganda.

CC 037.11.04.00.00. Drug Literature Index/ANTIINFECTIVE

AGENTS/Antiprotozoal drugs

CT EMTAGS: **etiology** (0135); epidemiology (0400); protozoon

(0751); Africa south of the Sahara (4032); therapy (0160);

controlled study (0197); clinical article (0152); **human**

(0888); virus (0761); infection (0310); ethnic or racial aspects

(0050); article (0060); priority journal (0007)

Medical Descriptors:

*malaria: ET, etiology

*malaria: EP, epidemiology

human immunodeficiency virus infection**: ET, etiologyhuman immunodeficiency virus infection**: EP, epidemiology***plasmodium falciparum**

*morbidity

uganda

Drug Descriptors:

*chloroquine: DT, drug therapy

*sulfadoxine: DT, drug therapy

*sulfadoxine: CB, drug combination

*pyrimethamine: DT, drug therapy

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blood transfusion
 fever
 drug efficacy
 antibody detection
 enzyme linked immunosorbent assay

parasitemia

drug response

***plasmodium falciparum**

parasite identification

Drug Descriptors:

*quinine: DT, drug therapy

RN 130-89-2; 130-95-0; 549-48-4; 7549-43-1

L94 ANSWER 76 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.

AN 91123111 EMBASE

TI The role of cytokines in malaria infection.

AU Maheshwari R.K.

CS Department of Pathology, Uniformed Services University of the Health Sciences, Bethesda, MD 20814-4799, United States

SO BULL. WHO, (1990) 68/SUPPL. (138-144).

ISSN: 0043-9686 CODEN: BWHOA6

CY Switzerland

DT Journal

FS 004 Microbiology

026 Immunology, Serology and Transplantation

030 Pharmacology

037 Drug Literature Index

LA English

AB We have tested the prophylactic effect of **Escherichia**

coli-derived recombinant human interferon gamma

(rHulFN-(.gamma.)) against sporozoite- or trophozoite-induced

Plasmodium cynomolgi B malaria infection in rhesus **monkeys**

. Data show that **treatment** with only five doses of

rHulFN-(.gamma.) (0.1 mg/kg body weight) given on days -2, 0, and +2 after infection protected the **monkeys** against

sporozoite-induced P. cynomolgi infection. Animals initially protected by rHulFN-(.gamma.) **treatment** remained

susceptible to reinfection. No inhibitory effect of rHulFN-(.gamma.)

was seen against trophozoite-induced infection. We have also tested

the effect of recombinant human tumour necrosis factor (rHuTNF) in

rhesus **monkeys**. No significant activity of TNF was seen

against trophozoite-induced P. cynomolgi B infection.

rHulFN-(.gamma.) inhibited schizogony in functional human

hepatocytes infected with **P. falciparum**

sporozoites. These results suggest that the inhibitory effect of IFN is limited to the exoerythrocytic stage of **parasite**

development. Interleukin-1 (IL-1) also inhibited hepatic development

of **P. falciparum** sporozoites; however, IL-1

treatment was effective only when applied before sporozoite

inoculation. IL-2 and TNF were effective in higher doses.

CT EMTAGS: infection (0310); prevention (0165); invertebrate (0723);

protozoon (0751); **monkey** (0725); mammal (0738); therapy

(0160); diagnosis (0140); nonhuman (0777); animal experiment (0112);

priority journal (0007); conference paper (0061)

Medical Descriptors:

*malaria: PC, prevention

*plasmodium cynomolgi

*sporozoite

*trophozoite

*immunity

monkey

prophylaxis

provocation test

nonhuman

further treatment of any kind. During this time, the patients remained clinically well. An additional six **HIV**-positive patients were **treated** with **malariotherapy** and have remained clinically well during the first 6 months after treatment. These initial studies demonstrate **malariotherapy** results in an increase in CD4 counts of HIV-positive patients. Furthermore, these increases persist beyond the presence of malaria, for at least 2 years.

ST RESEARCH ARTICLE; **PLASMODIUM VIVAX**; **HUMAN**
; **HIV**; **HUMAN IMMUNODEFICIENCY VIRUS**; **PARASITE**;
HOST; PATIENT; PATHOGEN; MALARIA; **MALARIOOTHERAPY**; CD4
COUNT; INFECTION; CLINICAL IMMUNOLOGY; **PARASITIC DISEASE**;
BLOOD AND LYMPHATIC DISEASE; THERAPEUTIC METHOD
CC Pathology, General and Miscellaneous-Therapy *12512
Immunology and Immunochemistry-Immunopathology, Tissue Immunology
*34508
Medical and Clinical Microbiology-Virology *36006
Parasitology-Medical *60504
Invertebrata, Comparative and Experimental Morphology, Physiology and
Pathology-Protozoa *64002
BC Retroviridae 02623
Sporozoa 35400
Hominidae 86215

L94 ANSWER 13 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1997:87570 HCAPLUS

DN 126:139350

TI Drug **treatment** of **HIV**-related opportunistic
infections

AU Klepser, Michael E.; Klepser, Teresa B.

CS Division of Clinical and Administrative Pharmacy, College of
Pharmacy, University of Iowa, Iowa City, IA, USA

SO Drugs (1997), 53(1), 40-73

CODEN: DRUGAY; ISSN: 0012-6667

PB Adis

DT Journal; General Review

LA English

CC 1-0 (Pharmacology)

AB A review with 178 refs. The AIDS epidemic has led to the emergence of several disease entities which in the pre-AIDS era were rare or seemingly innocuous. Experience of treating these diseases varies. In some instances, such as *Pneumocystis carinii* pneumonia, there is an abundance of published literature to direct our course of action. However, for many of these newly recognized diseases our treatment experience is limited. Furthermore, in many instances, well controlled trials evaluating treatment modalities in the AIDS population are lacking. We have identified 13 disease entities (*P. carinii* pneumonia, toxoplasmosis, cryptococcosis, histoplasmosis, **Mycobacterium tuberculosis**, *Mycobacterium avium* complex, cytomegalovirus, coccidioidomycosis, isosporiasis, candidosis, Kaposi's sarcoma, herpes simplex virus, and varicella zoster virus) and have reviewed the current literature with regard to their treatment.

ST review HIV infection

IT Infection

(**HIV**-related; drug **treatment** of **HIV**
-related opportunistic infections in **humans**)

IT **Human immunodeficiency virus 1**

(related infection; drug **treatment** of **HIV**
-related opportunistic infections in **humans**)

L94 ANSWER 14 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1997:160186 HCAPLUS

TI Computerized HIV and OI's information database systems

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id Parasite

*pyrimethamine: CB, drug combination
 RN 50-63-5; 54-05-7; 132-73-0; 3545-67-3; 2447-57-6; 58-14-0

L94 ANSWER 75 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 90335595 EMBASE
 TI Incidence of malaria and efficacy of oral quinine in patients recently infected with human **immunodeficiency** virus in Kinshasa, Zaire.
 AU Colebunders R.; Bahwe Y.; Nekwei W.; Ryder R.; Perriens J.; Nsimba K.; Turner A.; Francis H.; Lebughe I.; Van der Stuyft P.; Piot P.
 CS Projet SIDA, Department of Public Health, Kinshasa, Zaire
 SO J. INFECT., (1990) 21/2 (167-173).
 ISSN: 0163-4453 CODEN: JINFD2
 CY United Kingdom
 DT Journal
 FS 004 Microbiology
 006 Internal Medicine
 047 Virology
 LA English
 AB There is concern that the impaired cell mediated immunity caused by the human **immunodeficiency** virus may increase the risk or severity of **Plasmodium falciparum** infection and could lead eventually to a decreased response to standard antimalarial **treatment**. In 1986, at Mama Yemo Hospital, Kinshasa, Zaire, the incidence of malaria was determined in a cohort of 59 patients who had recently acquired **HIV-1** infection through blood transfusion and in a cohort of 83 **HIV-1** seronegative controls who were recipients of **HIV-1** seronegative blood. All cohort patients were asked to visit the study physician whenever they developed fever. On each of these occasions thick film was examined for the presence of malarial **parasites**. **HIV-1** seropositive patients presented more often with episodes of fever per person month observation than **HIV-1** seronegative patients ($P = 0.003$). The total number of positive thick films per person months observation was significantly higher among **HIV-1** seropositive patients than among the **HIV-1** seronegative ones, but percentages of positive thick films per episode of fever were the same in both groups (46%). During a 5 month period, cohort patients presenting with a moderate attack of malaria were **treated** with oral quinine 20 mg/kg daily in two doses for 5 days. Twenty-three (92%) of 25 **HIV-1** seropositive patients and 28 (82%) of 34 **HIV-1** seronegative patients had a negative film 7 days after starting **treatment**. This study suggests that there seems to be no direct interaction of major clinical importance between **HIV** infection and malaria.

CC 037.11.04.00.00. Drug Literature Index/ANTIINFECTIVE AGENTS/Antiprotozoal drugs
 CT EMTAGS: **diagnosis** (0140); therapy (0160); epidemiology (0400); virus (0761); Africa south of the Sahara (4032); child (0022); blood and hemopoietic system (0927); enzyme (0990); major clinical study (0150); controlled study (0197); **human** (0888); infection (0310); protozoon (0751); male (0041); female (0042); oral drug administration (0181); article (0060); priority journal (0007)
 Medical Descriptors:
 *malaria: DI, diagnosis
 *malaria: DT, drug therapy
 *malaria: EP, epidemiology
 *morbidity
 ***human immunodeficiency virus 1**
 ***human immunodeficiency virus infection**
 *zaire
 child

*quinine: DT, drug therapy
 *fansidar: DT, drug therapy
 RN 68583-22-2; 68583-29-9; 50-63-5; 54-05-7; 132-73-0; 3545-67-3;
 18323-44-9; 130-89-2; 130-95-0; 549-48-4; 7549-43-1; 37338-39-9

L94 ANSWER 32 OF 108 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 5
 AN 1996:61908 HCAPLUS
 DN 124:155745
 TI Liposome-mediated therapy of **human** immunodeficiency virus
 type-1 and Mycobacterium infections
 AU Duezguenes, Nejat; Flasher, Diana; Pretzer, Elizabeth; Konopka,
 Krystyna; Slepishkin, Vladimir A.; Steffan, Gerhard; Salem, Isam I.;
 Reddy, M. Venkata; Gangadharam, Pattisapu R.J.
 CS School of Dentistry, University of the Pacific, San Francisco, CA,
 94115, USA
 SO J. Liposome Res. (1995), Volume Date 1995, 5(4), 669-91
 CODEN: JLREE7; ISSN: 0898-2104
 DT Journal; General Review
 LA English
 CC 63-0 (Pharmaceuticals)
 AB A review, with 70 refs. on the authors recent work on the use of
 liposomes for the delivery of antiviral agents to **human**
 immunodeficiency virus type-1 (HIV-1) infected cells, and
 antimycobacterial drugs to cells harboring Mycobacterium avium
 complex or **Mycobacterium tuberculosis**. Sol. CD4
 has been used to target liposomes to HIV-1-infected cells.
 Antisense oligodeoxynucleotides have been effectively delivered into
 HIV-1-infected macrophages using pH-sensitive liposomes.
 PH-sensitive liposomes with serum stability are being developed as
 in vivo delivery vehicles. Liposomes encapsulating an **HIV**
 -1 protease **inhibitor** were more effective in inhibiting
 virus prodn. in infected macrophages than the free drug.
 ST review liposome bactericide Mycobacterium virucide HIV1
 IT Acquired immune deficiency syndrome
 Bactericides, Disinfectants, and Antiseptics
 Mycobacterium avium
Mycobacterium tuberculosis
 Tuberculostatics
 Virucides and Virustats
 (liposome-mediated therapy of HIV-1 and Mycobacterium infections)
 IT Virus, animal
 (human immunodeficiency 1, liposome-mediated therapy of
 HIV-1 and Mycobacterium infections)
 IT Pharmaceutical dosage forms
 (liposomes, liposome-mediated therapy of HIV-1 and Mycobacterium
 infections)

L94 ANSWER 33 OF 108 HCAPLUS COPYRIGHT 1998 ACS
 AN 1995:411658 HCAPLUS
 DN 122:182299
 TI Comparative complement selection in bacteria enables screening for
 lead compounds targeted to a purine salvage enzyme of parasites
 AU Eakin, Ann E.; Nieves-Alicea, Rene; Tosado-Acevedo, Rafael; Chin,
 Marian S.; Wang, Ching C.; Craig, Sydney P., III
 CS Sch. Med., Univ. Puerto Rico, San Juan, 00936-5067, P. R.
 SO Antimicrob. Agents Chemother. (1995), 39(3), 620-5
 CODEN: AMACQ; ISSN: 0066-4804
 DT Journal
 LA English
 CC 9-2 (Biochemical Methods)
 Section cross-reference(s): 10
 AB Expression plasmids encoding the hypoxanthine
 phosphoribosyltransferase (HPRTs) of Plasmodium **falciparum**
 , Schistosoma mansoni, Tritrichomonas foetus, and Homo sapiens were
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subcloned into genetically deficient *Escherichia coli* that requires complementation by the activity of the recombinant HPRT for growth on semidefined medium. Fifty-nine purine analogs were screened for their abilities to inhibit the growth of these bacteria. Several compds. that selectively altered the growth of the bacteria complemented by the malarial, schistosomal, or tritrichomonal HPRT compared with the growth of bacteria expressing the human enzyme were identified. These results demonstrate that the recombinant approach to screening compds. by complement selection in a comparative manner provides a rapid and efficient method for the identification of new lead compds. selectively targeted to the purine salvage enzymes of parasites.

ST parasite hypoxanthine phosphoribosyltransferase inhibitor screening *Escherichia*; *Plasmodium* hypoxanthine phosphoribosyltransferase inhibitor screening *Escherichia*; *Schistosoma* hypoxanthine phosphoribosyltransferase inhibitor screening *Escherichia*; *Tritrichomonas* hypoxanthine phosphoribosyltransferase inhibitor screening *Escherichia*

IT Antimalarials

Escherichia coli

Parasiticides

Plasmodium falciparum

Schistosoma mansoni

Tritrichomonas foetus

(screening in ***Escherichia coli*** for

inhibitors of a purine salvage enzyme of parasites)

IT 50-44-2 50-66-8 68-94-0 69-89-6 73-40-5 87-42-3 145-95-9
154-42-7 446-86-6, Azathioprine 767-69-1 1198-47-6
2036-13-7, 1H-Purine-6-carbonitrile 2545-26-8 10310-21-1
14225-97-9 14225-98-0 19447-73-5 19447-75-7 19690-23-4
20535-83-5 28128-41-8 37635-77-1 161746-77-6 161746-78-7
161746-79-8

RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(inhibitor of a purine salvage enzyme of parasites)

IT 9016-12-0

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(**inhibitors**; screening in ***Escherichia***

coli for **inhibitors** of a purine salvage enzyme of parasites)

L94 ANSWER 34 OF 108 MEDLINE

AN 96026495 MEDLINE

DN 96026495

TI Whole body hyperthermia associated with beta-carotene supplementation in patients with AIDS.

AU Pontiggia P; Bianchi Santamaria A; Alonso K; Santamaria L

CS C Golgi Institute of General Pathology, Centro Tumori, University of Pavia, Italy.

SO BIOMEDICINE AND PHARMACOTHERAPY, (1995) 49 (5) 263-5.

Journal code: A59. ISSN: 0753-3322.

CY France

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199602

AB The objective of this work was to check possible additive beneficial effects of whole body hyperthermia (WBH) associated with beta-carotene (BC) supplementation in patients with AIDS. In a pilot study, 10 HIV positive patients, (8 with AIDS and 2 with AIDS related complex, ARC), after AZT or DDI discontinuation, were first treated with one single session of WBH applied with a non-invasive procedure at 42 degrees C core temperature for one hour, and subsequently supplemented with BC 120 mg daily continuously. All

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Agents)

L94 ANSWER 31 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 95334702 EMBASE
 TI [Multiorganic failure in **Plasmodium falciparum** malaria].
 FALLO MULTIORGANICO EN EL PALUDISMO POR **PLASMODIUM FALCIPARUM**.
 AU Botella De Maglia J.; Cenicerros Rozalen I.; Oltra Chorda R.
 CS Unidad de Medicina Intensiva, Hospital La Fe, La Fe, Cuba
 SO Revista Clinica Espanola, (1995) 195/10 (688-692).
 ISSN: 0014-2565 CODEN: RCESA5
 CY Spain
 DT Journal
 FS 004 Microbiology
 037 Drug Literature Index
 LA Spanish
 SL Spanish; English
 AB A 44-year-old Spanish woman travelled in Kenya without doing correct malarial prophylaxis. Upon her return to Spain, she suffered from **Plasmodium falciparum** malaria. She was initially **treated** with chloroquine for three days, but her state worsened and she was admitted to our intensive care unit. On admission, **parasitaemia** was 22%. She had hyperpyrexia, obtundation, hypotension, tachycardia, tachypnoea, jaundice, digestive haemorrhage, petechiae in her soles, oliguria with elevation of serum uraemia and creatinine, anaemia, thrombocytopaenia, hypoproteinaemia, hyponatraemia, hypocalcaemia, metabolic acidosis and paramethers of disseminated intravascular coagulation. She was given quinine, sulfadoxine-pyrimethamine and clindamycin. An exchange transfusion was performed, during which an acute pulmonary oedema appeared, initially with high pulmonary artery wedge pressure. She required mechanical ventilation for 16 days and haemodialysis for 11 days. She remained in coma and had seizures which required diazepam, phenytoin and thiopentone. She received a total amount of 22 units of packed erythrocytes, 55 of platelets and 15 of plasma. After the first week, she had nosocomial infection due to **Escherichia coli**, Staphylococcus and Pseudomonas aeruginosa and was **treated** with the corresponding antibiotics. She cured completely. This case report gives us the possibility of discussing on frequent problems in the prevention and **treatment** of malaria, and on the **treatment** of severe, life-threatening malaria in the setting of the intensive care unit.
 CT EMTAGS: infection (0310); etiology (0135); therapy (0160); invertebrate (0723); protozoon (0751); organization and management (0142); bacterium (0762); mammal (0738); **human** (0888); case report (0151); female (0042); adult (0018); article (0060)
 Medical Descriptors:
 *malaria: ET, etiology
 *malaria: DT, drug therapy
plasmodium falciparum
 hospital infection
escherichia coli
 staphylococcus
 pseudomonas aeruginosa
human
 case report
 female
 adult
 article
 Drug Descriptors:
 *chloroquine: DT, drug therapy
 *clindamycin: DT, drug therapy

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0 (RNA, Viral)

L94 ANSWER 22 OF 108 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 2
 AN 1996:225320 HCAPLUS
 DN 124:306605
 TI The effect of thalidomide on the pathogenesis of **human**
 immunodeficiency virus type 1 and **M. tuberculosis**
 infection
 AU Klausner, Jeffrey D.; Makonkawkeyoon, Sanit; Akarasewi, Pasakorn;
 Nakata, Koh; Kasinrerker, Watchara; Corral, Laura; Dewar, Robin L.;
 Lane, H. Clifford; Freedman, Victoria H.; Kaplan, Gilla
 CS Medical Center, New York University, New York, USA
 SO J. Acquired Immune Defic. Syndr. Hum. Retrovirol. (1996), 11(3),
 247-57
 CODEN: JDSRET; ISSN: 1077-9450
 DT Journal
 LA English
 CC 1-5 (Pharmacology)
 AB Tumor necrosis factor alpha (TNF-.alpha.), a cytokine produced
 during the host defense against infection, is assocd. with fevers,
 weakness, and progressive wt. loss. Thalidomide inhibits the
 synthesis of TNF-.alpha. both in vitro and in vivo and may have
 clin. usefulness. The authors therefore initiated a pilot study of
 thalidomide **treatment** in patients with **human**
immunodeficiency virus type 1 (HIV-1)-assocd. wasting with
 or without concomitant infection with tuberculosis. Thirty-nine
 patients were randomly allocated to treatment with either
 thalidomide or placebo in a double-blind manner for 21 days.
 Thirty-two patients completed the study. In patients with
 concomitant HIV-1 and tuberculosis infections, thalidomide therapy
 was assocd. with a redn. in both plasma TNF-.alpha. levels and HIV-1
 levels. No significant redn. in either TNF-.alpha. or HIV-1 levels
 was obsd. in patients with HIV-1 infection only. During the study
 period, patients receiving thalidomide treatment showed a
 significant wt. gain (: 6.5%) relative to placebo-treated patients.
 Patients with simultaneous HIV-1 and tuberculosis infections
 experienced a higher mean wt. gain during thalidomide treatment than
 the group of patients with HIV-1 infection only. The results of
 this pilot study suggest that thalidomide may have a clin. role in
 enhancing wt. gain and possibly reducing TNF-.alpha. and HIV-1
 levels in patients with HIV-1 and concomitant mycobacterial
 infections.
 ST thalidomide HIV1 virus pathogenesis tuberculosis infection
 IT Tuberculosis
 (effect of thalidomide on pathogenesis of **human**
 immunodeficiency virus type 1 and **Mycobacterium**
tuberculosis infection in relation to tumor necrosis
 factor alpha prodn.)
 IT Virus, animal
 (human immunodeficiency 1, effect of thalidomide on
 pathogenesis of **human** immunodeficiency virus type 1 and
Mycobacterium tuberculosis infection in
 relation to tumor necrosis factor alpha prodn.)
 IT Lymphokines and Cytokines
 RL: BPR (Biological process); MFM (Metabolic formation); BIOL
 (Biological study); FORM (Formation, nonpreparative); PROC (Process)
 (tumor necrosis factor-.alpha., effect of thalidomide on
 pathogenesis of **human** immunodeficiency virus type 1 and
Mycobacterium tuberculosis infection in
 relation to tumor necrosis factor alpha prodn.)
 IT 50-35-1, Thalidomide
 RL: BAC (Biological activity or effector, except adverse); THU
 (Therapeutic use); BIOL (Biological study); USES (Uses)
 (effect of thalidomide on pathogenesis of **human**

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epidemiology (0400); mammal (0738); **human** (0888); major clinical study (0150); human tissue, cells or cell components (0111); infant (0014); child (0022); preschool child (0015); priority journal (0007); article (0060); adverse drug reaction (0198); iatrogenic disease (0300)

Medical Descriptors:

*anemia: DI, diagnosis
 *anemia: SI, side effect
 *malaria: DI, diagnosis
 *malaria: DT, drug therapy
 *malaria: ET, etiology

plasmodium falciparum

zaire

human immunodeficiency virus infection: CO, complication

blood transfusion

parasite isolation

hematocrit

treatment planning

antimalarial activity

nutrition

morbidity

human

major clinical study

human tissue

human cell

infant

preschool child

priority journal

article

Drug Descriptors:

*chloroquine: AE, adverse drug reaction
 *chloroquine: DT, drug therapy
 *chloroquine: PD, pharmacology

RN 50-63-5; 54-05-7; 132-73-0; 3545-67-3

L94 ANSWER 49 OF 108 CANCERLIT

AN 93306080 CANCERLIT

DN 93306080

TI Antitumor mechanisms of Z-100, an immunomodulatory arabinomannan X
 extracted from **Mycobacterium tuberculosis**: the importance of lymphocytes infiltrated into tumor sites.

AU Sasaki H; Schmitt D; Hayashi Y; Pollard R B; Suzuki F

CS Department of Internal Medicine, University of Texas Medical Branch, Galveston 77550.

SO NATURAL IMMUNITY, (1993). Vol. 12, No. 2, pp. 104-12.

Journal code: BGD. ISSN: 1018-8916.

DT Journal; Article; (JOURNAL ARTICLE)

FS MEDL; L; Priority Journals

LA English

OS MEDLINE 93306080

EM 199309

AB The mechanisms of increased host resistance to tumors following treatment with Z-100, an arabinomannan extracted from **Mycobacterium tuberculosis**, were investigated in mice bearing syngeneic solid tumors. When BALB/c mice bearing Meth-A solid tumors were treated intralesionally (i.l.) with a 10 mg/kg dose of Z-100, 74% of tumor growth was inhibited in the test group as compared with control mice treated with saline. However, no significant tumor inhibitory activity was observed when these mice were treated with various doses of Z-100 i.p. or i.v. In addition, tumor growth in X-irradiated mice (450 R, whole-body irradiation) and in mice treated with antilymphocyte serum was not suppressed even though Z-100 was administered into the tumor. The number of lymphocytes isolated from Z-100-treated tumor tissues increased

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nonhuman
controlled study
human cell
priority journal
article

RN 68583-22-2; 68583-38-0

L94 ANSWER 47 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS

AN 94:128790 BIOSIS

DN 97141790

TI Antibiotic sensitivity surveillance for the control of mycobacterial infections.

AU Fadda G

CS Ist. di Microbiol. e Virol. dell'Univ. degli Studi di Sassari, Viale San Pietro 43/B, 07100 Sassari, ITL

SO Igien. Moderna 99 (5). 1993. 632-655. ISSN: 0019-1655

LA Italian

AB With the increase in immunodeficiency virus (HIV) infection both in industrial and in developing countries, there has been a resurgence in tuberculosis (TB) and in infections due to non-tuberculous mycobacteria (NTM), mostly *M. avium-complex* (MAC). Since **M.**

tuberculosis is relatively virulent organism compared with other HIV-associated infections, TB is often the first (sentinel) infectious disease to appear in the setting of this progressive T-cell immunosuppression. When it is **treated** appropriately, the **HIV**-infected patient rarely dies from TB but from subsequent non-tuberculous infection (e.g. MAC). In the last two decades remarkable progress has been made in the treatment of TB mostly due to the better use of preexisting antitubercular drugs. Current protocols, which reintroduced the use of pyrazinamide, allowed to shortened the management of TB. However, when these regimens worked out under trial conditions were applied to field conditions, less favorable results were obtained. To further simplify therapy, improving compliance and to combat resistant mycobacteria and NTM, new antitubercular agents are needed. Various possibilities have emerged, such as the use of amikacin, quinolones, beta-lactamase

inhibitors associated with beta-lactam compounds and above all the new rifampycines. Conventional testing of mycobacterial susceptibility to antimicrobial drugs is based on growth/

inhibition of growth on solid medium (Lowenstein-Jensen, Ogawa, 7H10 or 7H11 agar). This approach provides a reasonable and satisfactory guideline for chemotherapy of tuberculosis. This method requires three or four weeks of incubation, cannot be used for testing of experimental drugs (for which the critical concentrations are not yet established), is not applicable to non tuberculous mycobacteria such as *M. avium-intracellulare*, and does not measure the degree of susceptibility of clinical isolates. To achieve these goals, alternative techniques based on broth cultures have been tried. Among these, the Bactec system for radiometric respirometry is the most widely used. This approach employs liquid media (Middlebrook 7H12) containing a ¹⁴C-labelled carbon source, palmitic acid, which when metabolized by bacteria yield detectable levels of ¹⁴CO₂. The amount of the ¹⁴CO₂ produced reflects the growth rate of mycobacteria. Susceptibility, that requires 4 to 5 days to report the results, is defined and a certain reduction (99%) of the metabolic activity of tested **M. tuberculosis** strain in a drug-containing vial compared to the unexposed control inoculated with a 1/100 dilution of the bacterial inoculum used for the drug-containing vials. In this report we discuss the pharmacological characteristics and "in vitro" antimycobacterial activity of all these drugs, some aspects related to the use of Bactec system, including qualitative and quantitative (MIC determination) drug susceptibility and interaction between drug combinations.

ST JOURNAL ARTICLE; MYCOBACTERIUM AVIUM; MYCOBACTERIUM

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TUBERCULOSIS; HUMAN; PYRAZINAMIDE;
 ANTIBACTERIAL-DRUG; AMIKACIN; ANTIBACTERIAL-DRUG; RIFAMPICIN;
 ANTIBACTERIAL-DRUG; THERAPEUTIC EFFICACY; **HUMAN**
 IMMUNODEFICIENCY VIRUS; OPPORTUNISTIC INFECTION

RN 98-96-4 (PYRAZINAMIDE)
 13292-46-1 (RIFAMPICIN)
 37517-28-5 (AMIKACIN)

CC Biochemical Studies-General 10060
 Pathology, General and Miscellaneous-Therapy *12512
 Pharmacology-Clinical Pharmacology *22005
 Immunology and Immunochemistry-Bacterial, Viral and Fungal *34504
 Immunology and Immunochemistry-Immunopathology, Tissue Immunology
 *34508
 Medical and Clinical Microbiology-Bacteriology *36002
 Medical and Clinical Microbiology-Virology *36006
 Chemotherapy-Antibacterial Agents *38504

BC Retroviridae 02623
 Mycobacteriaceae 08881
Hominidae 86215

L94 ANSWER 48 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 93115321 EMBASE
 TI **Plasmodium falciparum**-associated anemia in
 children at a large urban hospital in Zaire.

AU Hedberg K.; Shaffer N.; Davachi F.; Hightower A.; Lyamba B.; Paluku
 K.M.; Nguyen-Dinh P.; Breman J.G.

CS Malaria Branch F-12, Centers for Disease Control, Atlanta, GA 30333,
 United States

SO AM. J. TROP. MED. HYG., (1993) 48/3 (365-371).
 ISSN: 0002-9637 CODEN: AJTHAB

CY United States
 DT Journal
 FS 004 Microbiology
 037 Drug Literature Index
 038 Adverse Reactions Titles

LA English
 SL English

AB Chloroquine-resistant **Plasmodium falciparum**
 malaria and human **immunodeficiency virus (HIV)**
 infection through blood transfusions used to **treat**
 malaria-associated anemia are causes of increasing morbidity and
 mortality among children in Africa. To evaluate the role of malaria
 and other risk factors for pediatric anemia, we conducted a study of
 children brought to the emergency ward of a large urban hospital in
 Kinshasa, Zaire. A total of 748 children ages six through 59 months
 were enrolled; 318 (43%) children were anemic (hematocrit < 33%),
 including 74 (10%) who were severely anemic (hematocrit < 20%).
Plasmodium falciparum parasites were
 detected in 166 children (22%); hematocrits for these children (mean
 25.8%) were significantly lower than for aparasitemic children (mean
 33.7%; $P < 10^{-6}$). Fever with splenomegaly (odds ratio [OR] = 6.5, P
 = 0.02), **parasitemia** (OR = 3.5, $P < 0.001$), lower
 socioeconomic status (OR = 2.0, $P = 0.004$), and malnutrition (OR =
 1.8, $P = 0.06$) were independently associated with anemia in a
 multivariate model. Recent antimalarial therapy was also associated
 with a lower hematocrit, suggesting that chloroquine may have
 aggravated the anemia. A reassessment of the effectiveness of
 strategies to diagnose and **treat** malaria and malnutrition
 is necessary to decrease the high prevalence of anemia and the
 resultant high rate of blood transfusions in areas endemic for
 malaria and **HIV**.

CT EMTAGS: **diagnosis** (0140); **infection** (0310);
 therapy (0160); etiology (0135); invertebrate (0723); protozoon
 (0751); Africa (0403); Africa south of the Sahara (4032);

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- inhibition with)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, dithiophosphate-linked, infection by pathogen inhibition with)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, phosphoramidate-linked, infection by pathogen inhibition with)
- IT Microorganism
(pathogenic, infection by, oligonucleotides for inhibition of)
- IT Anthelmintics
(schistosomicides, oligonucleotides inhibiting replication or reprod. of Schistosoma)
- IT 146416-16-2 150875-86-8 150875-87-9
RL: BIOL (Biological study)
(antimalarial)
- IT 37228-74-3, Exonuclease
RL: BIOL (Biological study)
(antimalarial oligonucleotide resistant to degradn. by)
- IT 9031-61-2
RL: BIOL (Biological study)
(dihydrofolate reductase-, gene for, of Plasmodium **falciparum**, oligonucleotide inhibiting)
- IT 54-05-7, Chloroquine 56-54-2, Quinidine 58-14-0, Pyrimethamine
130-95-0, Quinine 53230-10-7, Mefloquine
RL: BIOL (Biological study)
(malarial pathogen resistant to, oligonucleotide inhibiting)
- IT 146416-19-5
RL: BIOL (Biological study)
(oligonucleotide complementary to first nucleotides of gene P195, for inhibition of Plasmodium **falciparum**)
- IT 150875-88-0
RL: BIOL (Biological study)
(oligonucleotide complementary to first nucleotides of gene P195, Plasmodium **falciparum** inhibition with)
- IT 146416-15-1 146416-15-1D, phosphoroamidate and phosphorodithioate and phosphorothioate derivs.
RL: BIOL (Biological study)
(oligonucleotide complementary to first nucleotides of gene for dihydrofolate reductase-thymidylate synthase, for inhibition of Plasmodium **falciparum**)
- IT 150875-91-5 150875-92-6
RL: BIOL (Biological study)
(oligonucleotide complementary to first nucleotides of gene for dihydrofolate reductase-thymidylate synthase, Plasmodium **falciparum** inhibition with)
- IT 146416-14-0 146416-14-0D, phosphoroamidate and phosphorodithioate and phosphorothioate derivs.
RL: BIOL (Biological study)
(oligonucleotide complementary to nucleotides of gene P195, for inhibition of Plasmodium **falciparum**)
- IT 150875-89-1 150875-90-4
RL: BIOL (Biological study)
(oligonucleotide complementary to nucleotides of gene P195, Plasmodium **falciparum** inhibition with)
- IT 146416-19-5D, phosphoroamidate and phosphorodithioate and phosphorothioate derivs.
RL: BIOL (Biological study)
(oligonucleotides complementary to first nucleotides of gene P195, for inhibition of Plasmodium **falciparum**)
- IT 9002-03-3
RL: BIOL (Biological study)
(thymidylate synthase-, gene for, of Plasmodium

falciparum, oligonucleotide inhibiting)

L94 ANSWER 46 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 94030068 EMBASE
 TI Reduced microbicidal and anti-tumour activities of human monocytes after ingestion of **Plasmodium falciparum** -infected red blood cells.
 AU Fiori P.L.; Rappelli P.; Mirkarimi S.N.; Ginsburg H.; Cappuccinelli P.; Turrini F.
 CS Department of Biological Chemistry, Institute of Life Sciences, Hebrew University, Jerusalem 91904, Israel
 SO PARASITE IMMUNOL., (1993) 15/12 (647-655).
 ISSN: 0141-9838 CODEN: PAIMD8
 CY United Kingdom
 DT Journal
 FS 004 Microbiology
 016 Cancer
 025 Hematology
 026 Immunology, Serology and Transplantation
 LA English
 SL English
 AB Oxidatively stressed red blood cells (RBC) and **Plasmodium falciparum**- infected RBC (PRBC) are avidly phagocytosed by human peripheral monocytes. Following the ingestion of PRBC the monocytes' ability to phagocytose PRBC and to generate aggressive oxidative compounds is severely impaired. In the present work the microbicidal and anti-tumour capacities of monocytes fed with diamide-treated RBC and PRBC harbouring mature (trophozoite) **parasites** have been investigated. The capacity of the latter, but not of the former, to phagocytose **Escherichia coli** and Staphylococcus aureus and to kill them, as well as ingested Candida albicans cells intracellularly, was found to be markedly impaired. Monocytes that have ingested PRBC had a significantly reduced cytostatic and cytolytic activities against a lymphoblastic tumour cell line. Monocytes fed with oxidatively stressed RBC had normal or sometimes even greater anti-tumour activities. Monocytes that have ingested PRBC showed a reduced capability to produce superoxide following stimulation with phorbol ester. Such impairment in monocyte functions may explain the reduced antibacterial and anti-tumour activities of monocytes in malaria patients, and could be consequential to their ability to resist bacterial infections and to provide means for the control of tumour development in those patients.
 CT EMTAGS: **invertebrate** (0723); protozoon (0751); **reticuloendothelial system** (0924); blood and hemopoietic system (0927); infection (0310); etiology (0135); plant (0699); fungus (0763); bacterium (0762); mammal (0738); **human** (0888); nonhuman (0777); controlled study (0197); human tissue, cells or cell components (0111); priority journal (0007); article (0060)
 Medical Descriptors:
 ***plasmodium falciparum**
 *monocyte
 *bactericidal activity
 *antineoplastic activity
 *malaria: ET, etiology
 erythrocyte
 candida albicans
 phagocytosis
host parasite interaction
escherichia coli
 staphylococcus aureus
human

Toxoplasma
 Trichinella spiralis
 Trichomonas
 (drug-resistant, treatment of, with antisense oligonucleotides)
 IT Leishmania
 Malaria
 Parasite
 Plasmodium **falciparum**
 Schistosoma
 Trypanosoma
 Virus
 (infection by, oligonucleotides for inhibition of)
 IT Gene, microbial
 RL: BIOL (Biological study)
 (oligonucleotide hybridizing with vital, of pathogen, for
 inhibiting infection by pathogen)
 IT Bacteria
 (oligonucleotides for inhibition of)
 IT Anti-infective agents
 (oligonucleotides for inhibition of pathogen for)
 IT Bactericides, Disinfectants, and Antiseptics
 (oligonucleotides inhibiting replication or reprod. of bacteria)
 IT Antimalarials
 (oligonucleotides inhibiting replication or reprod. of malaria
 pathogen)
 IT Parasiticides
 (oligonucleotides inhibiting replication or reprod. of parasite)
 IT Virucides and Virustats
 (oligonucleotides inhibiting replication or reprod. of virus)
 IT Trypanosomicides
 (oligonucleotides inhibiting replication or reprod. of
 Trypanosoma)
 IT Pharmaceuticals
 (pathogens resistant to, treatment of, with antisense
 oligonucleotides)
 IT Intestine, disease
 (amebiasis, drug-resistant, treatment of, with antisense
 oligonucleotides)
 IT Mycosis
 (blasto-, drug-resistant, treatment of, with antisense
 oligonucleotides)
 IT Therapeutics
 (chemo-, pathogen resistant to, oligonucleotide inhibiting)
 IT Mycosis
 (coccidioido-, drug-resistant, treatment of, with antisense
 oligonucleotides)
 IT Skin, disease
 (dermatophytosis, drug-resistant, treatment of, with antisense
 oligonucleotides)
 IT Therapeutics
 (geno-, infection by pathogen inhibition by, oligonucleotides
 for)
 IT Intestine, disease
 (giardiasis, drug-resistant, treatment of, with antisense
 oligonucleotides)
 IT Venereal disease
 (lymphogranuloma venereum, drug-resistant, infection with,
 treatment of, with antisense oligonucleotides)
 IT Nucleotides, polymers
 RL: BIOL (Biological study)
 (oligo-, infection by pathogen inhibition with)
 IT Nucleotides, polymers
 RL: BIOL (Biological study)
 (oligo-, deoxyribo-, thiophosphate-linked, infection by pathogen

parasite merozoite

RL: PRP (Properties)

(amino acid sequence of, prophylaxis and **treatment** of
HIV infection with)

L94 ANSWER 45 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1993:617377 HCAPLUS
DN 119:217377
TI Antiparasitic oligonucleotides active against drug-resistant malaria
IN Rapaport, Eliezer; Zamecnik, Paul C.
PA Worcester Foundation for Experimental Biology, USA
SO PCT Int. Appl., 47 pp.
CODEN: PIXXD2
PI WO 9313740 A2 930722
DS W: CA, JP, KR, US
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
AI WO 92-US11202 921231
PRAI US 91-815393 911231
DT Patent
LA English
IC ICM A61K031-70
ICS C12N015-11
CC 1-5 (Pharmacology)
Section cross-reference(s): 3
AB Active infection by a pathogen, esp. *Plasmodium falciparum*
, is inhibited by administering an oligonucleotide that inhibits the
replication or reprod. of the pathogen. Materials and methods are
provided for antisense oligonucleotide therapy against
drug-resistant or -sensitive pathogens. Phosphorothioate 5'-GTC GCA
GAC TTG TTC CAT CAT-3' (I, complementary to the 1st 21 nucleotides
of the open reading frame of *P. falciparum* dihydrofolate
reductase-thymidylate synthase gene starting with the start codon),
with the last 3' phosphodiester bond being a phosphorbutylamidate
for inhibition of exonuclease activity, was equally effective in
inhibiting the growth and invasion of chloroquine-resistant and
-sensitive strains of *P. falciparum*. I had higher
antimalarial activity than an oligonucleotide of identical sequence
but lacking the Bu phosphoramidate group at the 3' end.
ST antisense oligonucleotide therapy pathogen; drug resistant malaria
antisense oligonucleotide therapy; *Plasmodium* gene inhibition
oligonucleotide
IT Trypanosoma cruzi
(Chagas' disease from, drug-resistant, treatment of, with
antisense oligonucleotides)
IT Gene, microbial
RL: BIOL (Biological study)
(P195, of *Plasmodium falciparum*, antimalarial
oligonucleotides hybridizing with)
IT Candida
Cestode
Chlamydia trachomatis
Cryptococcus (fungus)
Histoplasma capsulatum
Nematode
Pneumocystis carinii
(drug-resistant, infection with, **treatment** of, with
antisense oligonucleotides)
IT Ascaris
Aspergillus
Cryptosporidium
Filaria
Rickettsia prowazekii
Rocky Mountain spotted fever
Sporotrichum

- cerebrospinal fluid with cell cycle phase-specific therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Nervous system
(disease, neurol. disorder treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Virus, animal
(human T-cell leukemia type I, neurol. virus infection treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Virus, animal
(human T-cell leukemia type II, neurol. virus infection treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Virus, animal
(human immunodeficiency 1, neurol. virus infection **treatment** using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Virus, animal
(human immunodeficiency 2, neurol. virus infection **treatment** using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Virus, animal
(lenti-, neurol. virus infection treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Pharmaceutical dosage forms
(liposomes, neurol. disorder treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Neoplasm inhibitors
(metastasis, neurol. disorder treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Virus, animal
(retro-, neurol. virus infection treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Virus, animal
(slow, neurol. virus infection treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Neoplasm inhibitors
(subarachnoid space, metastasis, neurol. disorder treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT Meninges
(subarachnoid space, neoplasm, metastasis, inhibitors, neurol. disorder treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT 147-94-4, Cytarabine
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(neurol. disorder treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
- IT 50-99-7, Glucose, biological studies 57-50-1, Sucrose, biological studies 99-20-7, Trehalose 31112-62-6, Metrizamide 66108-95-0, Iohexol 92339-11-2, Iodixanol

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(neurol. disorder treatment using administration to cerebrospinal
fluid with therapeutic dispersion allowing persistence in
cerebro-ventricular space)

IT 50-02-2, Dexamethasone
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oral dexamethasone redn. of toxicity of ara-C dispersion
intrathecal and intraventricular treatment in cancer patients
with neoplastic meningitis)

L94 ANSWER 39 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1994:672192 HCAPLUS
DN 121:272192
TI Pharmaceutical tryptophan-containing dipeptide compositions and use
in treatment of a variety of diseases
IN Khavinson, Vladimir Khatskelevi; Morozov, Vyacheslav Grigorievic;
Sery, Sergiy Vladimirovich; Green, Lawrence; Sinackevich, Nicolay V.;
Kozhemyakin, Andrei L.
PA Cytoven International N.V., USA
SO PCT Int. Appl., 117 pp.
CODEN: PIXXD2
PI WO 9420063 A2 940915
DS W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, HU,
JP, KP, KR, KZ, LK, LU, LV, MG, MN, MW, NL, NO, NZ, PL, PT, RO,
RU, SD, SE, SI, SK, UA, US, UZ, VN
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR,
IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG

AI WO 94-US2354 940304
PRAI US 93-26341 930304
DT Patent
LA English
IC ICM A61K
CC 1-12 (Pharmacology)
Section cross-reference(s): 8, 14, 15, 63

AB The present invention provides compns. and methods for treatment of
a variety of disease states. The methods generally comprise
administering to a host a therapeutically effective amt. of a
dipeptide having the formula X-Trp or a pharmaceutically acceptable
salt thereof, wherein X is glutamine, glutamate, leucine, or
isoleucine. The present invention is useful for **treatment**
of infections, hyperimmune states, **immunodeficiencies**, and
the like. Bronchial asthma patients, patients infected with
Shigella dysentery, pregnant women, etc. were treated with Ile-Trp.
People exposed to radiation at Chernobyl were treated with Glu-Trp.

ST tryptophan dipeptide pharmaceutical; infection treatment tryptophan
dipeptide; immune system tryptophan dipeptide; disease treatment
tryptophan dipeptide; radiation tryptophan dipeptide

IT Dysentery
(Shigella; pharmaceutical tryptophan-contg. dipeptide compns. and
use in treatment of variety of diseases)

IT Shigella
(dysentery; pharmaceutical tryptophan-contg. dipeptide compns.
and use in treatment of variety of diseases)

IT Cosmetics
(fewer allergy reactions to; pharmaceutical tryptophan-contg.
dipeptide compns. and use in treatment of variety of diseases)

IT Anesthetics
Anti-infective agents
Neoplasm inhibitors
(in tryptophan-contg. dipeptide compns.; pharmaceutical
tryptophan-contg. dipeptide compns. and use in treatment of
variety of diseases)

IT Interferons
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(in tryptophan-contg. dipeptide compns.; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)

- IT Bacteria
 - Candida albicans
 - Fungi
 - Histoplasma capsulatum
 - Leishmania
 - Mycobacterium leprae
 - Mycobacterium tuberculosis**
 - Mycobacterium
 - Parasite**
 - Plasmodium (malarial genus)
 - Virus, animal
 - (infection; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Staphylococcus aureus
 - (peritonitis from methicillin-resistant; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Acne
 - Acquired immune deficiency syndrome
 - Allergy inhibitors
 - Asthma
 - Bactericides, Disinfectants, and Antiseptics
 - Burn
 - Common cold
 - Dentifrices
 - Eye, disease
 - Fungicides and Fungistats
 - Immunity
 - Immunodeficiency
 - Immunostimulants
 - Leprosy
 - Parasiticides**
 - Parturition
 - Pharmaceutical dosage forms
 - Pregnancy
 - Psoriasis
 - Radiation sickness
 - Skin, disease
 - Toxemia of pregnancy
 - Tuberculosis
 - Virucides and Virustats
 - Wound healing promoters
 - (pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Blood transfusion
 - (prevention of alloblood rejection after; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Transplant and Transplantation
 - (prevention of rejection of; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Antibiotics
 - RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 - (pyrazinamide, in tryptophan-contg. dipeptide compns.; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Malaria
 - (relapsing forms of; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Staphylococcus
 - (skin disease from antibiotic-resistant; pharmaceutical

- tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Aspergillus
(aspergillosis from, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Mycosis
(blastomycosis, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Candida
(candidiasis from, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Inflammation
(cellulitis, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Therapeutics
(chemotherapy, complications and side effects from; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Skin, disease
(chromomycosis, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Osteomyelitis
(chronic, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Mycosis
(coccidioidomycosis, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Temperature effects, biological
(cold, frostbite, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Intestine, disease
(colon, infection, bacterial; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Cryptococcus neoformans
(cryptococcosis from, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Virus, animal
(dengue, infection; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Peptides, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(di-, tryptophan-contg.; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Gingiva
(disease, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Respiratory tract
(disease, acute, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Tooth
(disease, caries, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Ear
(disease, infection, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Lymphatic system
(disease, inflammation, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Peritoneum
(disease, peritonitis, from methicillin-resistant Staphylococcus aureus; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Prostate gland
(disease, prostatitis, pharmaceutical tryptophan-contg. dipeptide

comps. and use in treatment of variety of diseases)

IT Sinus
(disease, sinusitis, pharmaceutical tryptophan-contg. dipeptide
comps. and use in treatment of variety of diseases)

IT Hair
(follicle, disease, inflammation, pharmaceutical
tryptophan-contg. dipeptide comps. and use in treatment of
variety of diseases)

IT Bone, disease
(fracture, pharmaceutical tryptophan-contg. dipeptide comps. and
use in treatment of variety of diseases)

IT Skin, disease
(furunculosis, pharmaceutical tryptophan-contg. dipeptide comps.
and use in treatment of variety of diseases)

IT Transplant and Transplantation
(graft-vs.-host reaction, pharmaceutical tryptophan-contg.
dipeptide comps. and use in treatment of variety of diseases)

IT Virus, animal
(hepatitis, infection; pharmaceutical tryptophan-contg. dipeptide
comps. and use in treatment of variety of diseases)

IT Virus, animal
(herpes, infection; pharmaceutical tryptophan-contg. dipeptide
comps. and use in treatment of variety of diseases)

IT Virus, animal
(**human** immunodeficiency, infection; pharmaceutical
tryptophan-contg. dipeptide comps. and use in treatment of
variety of diseases)

IT Bone, disease
Kidney, disease
Lung, disease
Stomach, disease
(infection, bacterial; pharmaceutical tryptophan-contg. dipeptide
comps. and use in treatment of variety of diseases)

IT Virus, animal
(influenza, infection; pharmaceutical tryptophan-contg. dipeptide
comps. and use in treatment of variety of diseases)

IT Lymphokines and Cytokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(interleukins, in tryptophan-contg. dipeptide comps.;
pharmaceutical tryptophan-contg. dipeptide comps. and use in
treatment of variety of diseases)

IT Neoplasm inhibitors
(leukemia, in tryptophan-contg. dipeptide comps.; pharmaceutical
tryptophan-contg. dipeptide comps. and use in treatment of
variety of diseases)

IT Mycosis
(mucormycosis, pharmaceutical tryptophan-contg. dipeptide comps.
and use in treatment of variety of diseases)

IT Mammary gland
(neoplasm, radiotherapy-treated; pharmaceutical tryptophan-contg.
dipeptide comps. and use in treatment of variety of diseases)

IT Blastomyces brasiliensis
(paracoccidioidomycosis from, pharmaceutical tryptophan-contg.
dipeptide comps. and use in treatment of variety of diseases)

IT Kidney, disease
(pyelonephritis, pharmaceutical tryptophan-contg. dipeptide
comps. and use in treatment of variety of diseases)

IT Skin, disease
(pyoderma, pharmaceutical tryptophan-contg. dipeptide comps. and
use in treatment of variety of diseases)

IT Intestine, disease
(small, infection, bacterial; pharmaceutical tryptophan-contg.
dipeptide comps. and use in treatment of variety of diseases)

IT Sporothrix schenckii

- (sporotrichosis from, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Animal growth regulators
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(transforming growth factors, in tryptophan-contg. dipeptide compns.; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Skin
(transplant, prevention of rejection of; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Lymphokines and Cytokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(tumor necrosis factor, in tryptophan-contg. dipeptide compns.; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Immunization
(vaccination, augmentation of; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Virus, animal
(varicella-zoster, herpes zoster from, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT Acne
(vulgaris, pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT 98-96-4, Pyrazinamide
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antibiotics, in tryptophan-contg. dipeptide compns.; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT 54-85-3, Isoniazid 57-92-1, Streptomycin, biological studies
69-53-4, Ampicillin 80-08-0 1397-89-3, Amphotericin B
2022-85-7, Flucytosine 2030-63-9, Clofazimine 13292-46-1,
Rifampin 62683-29-8D, Colony-stimulating factor, compds.
65277-42-1, Ketoconazole 84625-61-6, Itraconazole 86386-73-4,
Fluconazole
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(in tryptophan-contg. dipeptide compns.; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT 61-32-5, Methicillin
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(peritonitis from Staphylococcus aureus resistant to; pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT 13589-06-5, Ile-Trp 38101-59-6
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)
- IT 5156-22-9, Leu-Trp 66851-83-0
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pharmaceutical tryptophan-contg. dipeptide compns. and use in treatment of variety of diseases)

L94 ANSWER 40 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1994:144158 HCAPLUS

DN 120:144158

TI Nuclease-resistant oligonucleotides stabilized by internal hybridization and their use as therapeutic agents

IN Agrawal, Sudhir; Tang, Jin Yan

PA Hybridon, Inc., USA

KATHLEEN FULLER BT/LIBRARY 308-4290

SO PCT Int. Appl., 48 pp.
CODEN: PIXXD2

PI WO 9401550 A1 940120

DS W: AT, AU, BB, BG, BR, CA, CH, CZ, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MG, MN, MW, NL, NO, NZ, PL, RO, RU, SD, SE, SK, UA, US, VN
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG

AI WO 93-US6326 930702

PRAI US 92-909069 920702

DT Patent

LA English

IC ICM C12N015-11
ICS C07H021-00; A61K031-70

CC 63-5 (Pharmaceuticals)

AB Improved antisense oligonucleotides that are resistant to nucleolytic degradn. have two regions: a target hybridizing region complementary to a nucleic acid sequence that is from a pathogen, or a cellular gene; and a self-complementary region. Such oligonucleotides are called self-stabilized oligonucleotides. The nuclease resistance of these oligonucleotides may be increased by using unusual bondings such as phosphorothioates. An oligonucleotide complementary to the gag gene of HIV-1 was digested by snake venom phosphodiesterase with a half-life of 75 s; a self-stabilized oligonucleotide carrying a 3' tail of 10 self-complementary oligonucleotides had a half-life of 950 s under the same conditions. The nuclease resistance of these oligonucleotides was greatly increased in the phosphorothioate analog; the half-life of the analog of the first oligonucleotides was increased to 4 h and the analog of the second was essentially undegraded after 4 h. The self-stabilized oligonucleotide was an effective **inhibitor** of **HIV-1** growth in H9 lymphocytes, as judged by inhibition of p24 synthesis, with an IC50 of 0.25-0.35 .mu.g/mL, compared to 2-2.8 .mu.g/mL for the non-stabilized oligonucleotide.

ST oligonucleotide self stabilized antisense therapeutic; HIV gag gene antisense oligonucleotide selfstabilized

IT Fasciola hepatica
Leishmania
Plasmodium **falciparum**
Trypanosoma brucei
Virus, plant
(infection by, treatment of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)

IT Ribozymes
RL: BIOL (Biological study)
(inhibition of gene expression with nucleolysis-resistant, stabilization by internal hybridization of)

IT Gene, animal
RL: BIOL (Biological study)
(oligonucleotides for inhibition of expression of, stabilization against nucleolysis by internal hybridization of)

IT Virus, animal
(oligonucleotides for treatment of infection by, stabilization against nucleolysis by internal hybridization of)

IT Glycolipoproteins
RL: BIOL (Biological study)
(PrP (prion protein), gene for, inhibition of expression of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)

IT Glycoproteins, specific or class
RL: BIOL (Biological study)
(amyloid A4, pre-, gene for, inhibition of expression of,

- oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)
- IT Deoxyribonucleic acids
RL: BIOL (Biological study)
(complementary, antisense, oligonucleotides, therapeutic, self-stabilized, internal hybridization in, for stabilization against nucleolysis)
- IT Virus, plant
(cucumo-, infection by, treatment of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)
- IT Virus, animal
(foot-and-mouth disease, infection by, treatment of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)
- IT Virus, animal
(herpes simplex, infection by, treatment of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)
- IT Virus, animal
(human **immunodeficiency** 1, infection by, **treatment** of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)
- IT Virus, animal
(human papilloma, infection by, treatment of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)
- IT Virus, animal
(influenza, infection by, treatment of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, self-stabilized, internal hybridization in, for stabilization against nucleolysis)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, alkylphosphonate-linked, in self-stabilized oligonucleotides showing internal hybridization in, for stabilization against nucleolysis)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, alkylphosphonothioate-linked, in self-stabilized oligonucleotides showing internal hybridization in, for stabilization against nucleolysis)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, dithiophosphate-linked, self-stabilized, internal hybridization in, for stabilization against nucleolysis)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, phosphoramidate-linked, in self-stabilized oligonucleotides showing internal hybridization in, for stabilization against nucleolysis)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, phosphotriester-linked, in self-stabilized oligonucleotides showing internal hybridization in, for stabilization against nucleolysis)
- IT Nucleotides, polymers
RL: BIOL (Biological study)
(oligo-, thiophosphate-linked, self-stabilized, internal hybridization in, for stabilization against nucleolysis)
- IT Microorganism

(pathogenic, oligonucleotides for treatment of infection by, stabilization against nucleolysis by internal hybridization of)

IT Gene
 RL: BIOL (Biological study)
 (transforming, inhibition of expression of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)

IT Virus, animal
 (varicella-zoster, infection by, treatment of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)

IT Virus, animal
 (yellow fever, infection by, treatment of, oligonucleotides for, nucleolysis-resistant, stabilization by internal hybridization of)

L94 ANSWER 41 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 94142324 EMBASE
 TI The resolution of acute malaria in a definitive model of B cell deficiency, the J(H)D mouse.
 AU Van der Heyde H.C.; Huszar D.; Woodhouse C.; Manning D.D.; Weidanz W.P.
 CS Med. Microbiology/Immunology Dept., University of Wisconsin, 1300 University Avenue, Madison, WI 53706, United States
 SO J. IMMUNOL., (1994) 152/9 (4557-4562).
 ISSN: 0022-1767 CODEN: JOIMA3
 CY United States
 DT Journal
 FS 004 Microbiology
 026 Immunology, Serology and Transplantation
 LA English
 SL English
 AB Because the role of cell-mediated immunity (CMI) in the resolution of blood-stage malaria remains unclear, we examined the question of whether **mice** completely lacking Ab-mediated immunity (AMI) but possessing some CMI can resolve experimental malaria previously reported not to require AMI for resolution. Severe combined **immunodeficient mice** reconstituted with enriched immune T cells (<0.5% B220+ cells) suppressed acute Plasmodium chabaudi adami **parasitemia**, suggesting that T, but not B, cells are required to clear this form of malaria. In addition, J(H)D **mice**, which are a definitive model of B cell deficiency, were also shown to resolve P. chabaudi adami, Plasmodium vinckei petteri and Plasmodium chaubadi chabaudi malaria. These observations collectively establish that CMI alone can mediate the clearance of acute malaria caused by these subspecies of Plasmodium. Moreover, the protective cell-mediated immune response involved depends upon CD4+ T cells because J(H)D **mice treated** with anti-CD4 mAb do not resolve their infections. These results suggest that evaluation of immunization regimens to activate CD4+ T cell dependent cell mediated immunity against **Plasmodium falciparum** may be appropriate.

CT EMTAGS: infection (0310); etiology (0135); blood and hemopoietic system (0927); lymphatic system (0929); invertebrate (0723); protozoon (0751); therapy (0160); prevention (0165); nonhuman (0777); female (0042); mouse (0727); mammal (0738); animal model (0106); biological model (0502); controlled study (0197); animal tissue, cells or cell components (0105); priority journal (0007); article (0060)
 Medical Descriptors:
 *malaria: ET, etiology
 *immune deficiency
 humoral immunity
 suppressor cell

plasmodium chabaudi
 plasmodium vinckei
 cellular immunity
 immunization
 b lymphocyte
 nonhuman
 female
 mouse
 animal model
 controlled study
 animal tissue
 animal cell
 priority journal
 article

L94 ANSWER 42 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 94321839 EMBASE
 TI Efficacy of Ro42-1611 (arteflene) in the **treatment** of
 patients with mild malaria: A clinical trial in Cameroon.
 AU Somo-Moyou R.; Mittelholzer M.-L.; Sorenson F.; Haller L.; Sturchler
 D.
 CS F. Hoffmann-La Roche Ltd, Dept POBT, CH-4002 Basel, Switzerland
 SO TROP. MED. PARASITOL., (1994) 45/3 (288-291).
 ISSN: 0177-2392 CODEN: TMPAEY
 CY Germany, Federal Republic of
 DT Journal
 FS 004 Microbiology
 007 Pediatrics and Pediatric Surgery
 017 Public Health, Social Medicine and Epidemiology
 037 Drug Literature Index
 038 Adverse Reactions Titles
 LA English
 SL English
 AB The novel antimalarial Ro 42-1611 (arteflene) was evaluated for
 safety and efficacy in an open, non-comparative study of patients
 with mild malaria in the south of Cameroon. Thirty male patients
 aged 12 to 42 years, with an initial **Plasmodium**
falciparum count of >5000 (mean: 21,406) **parasites**
 /.mu.l and a body temperature of 37.7° to 39.8.degree.C, were
 selected to receive a single dose of arteflene, corresponding to 25
 .+- 2.5 mg /kg bodyweight. Efficacy was assessed at 6, 9,12, 24,
 36, 48 and 72 hours, and at seven days by: reduction in
parasitaemia and time to **parasite** clearance;
 resolution of fever and clinical cure (defined as the absence of
 signs and symptoms of malaria). Adverse events were reported at
 baseline and at each assessment point, and laboratory tests were
 carried out at 2 and 7 days. The mean number of **parasites**
 /.mu.l fell from 21,406 at baseline to 157 after 48 hours, at which
 point 80% of patients were completely free of **parasites**.
 Mean body temperature was reduced from 38.9.degree.C at baseline to
 37.3.degree.C 12 hours after arteflene administration, and by this
 time 80% of patients had a normal temperature. Clinical cure rates
 were also high, with 70% of patients free of all signs and symptoms
 after 24 hours. However, by day 7, 6/30 (20%) presented with smears
 positive for **P. falciparum**. There were no
 adverse events considered to be related to **treatment**. A
 single dose of 25 mg/kg arteflene was found to be an effective and
 well-tolerated **treatment** for mild **P.**
falciparum malaria.
 CT EMTAGS: **Africa** (0403); Africa south of the Sahara (4032);
infection (0310); therapy (0160); invertebrate (0723);
 protozoon (0751); mammal (0738); **human** (0888); male
 (0041); clinical article (0152); adolescent (0017); school child
 (0016); child (0022); adult (0018); oral drug administration (0181);

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human experiment (0104); conference paper (0061); adverse drug reaction (0198); iatrogenic disease (0300)

Medical Descriptors:

*antimalarial activity

cameroon

malaria: DT, drug therapy

drug efficacy

drug safety

plasmodium falciparum

body temperature

time

typhoid fever: DT, drug therapy

typhoid fever: SI, side effect

human

male

clinical article

adolescent

school child

adult

oral drug administration

clinical trial

conference paper

Drug Descriptors:

*antimalarial agent: AE, adverse drug reaction

*antimalarial agent: CT, clinical trial

*antimalarial agent: DT, drug therapy

chloramphenicol: DT, drug therapy

RN 56-75-7; 134-90-7; 2787-09-9

L94 ANSWER 43 OF 108 AIDSLINE

AN 1993:11704 AIDSLINE

DN ICA9-93334739

TI Cerebral toxoplasmosis and cerebral tuberculosis simultaneously in an HIV + patient with median CD4 + counts of 372 cells/mm3 - 21%.

AU Oliveira M P; Silva L C; Castineiras T M; Martins L; Piloto J H; Peixoto C A

CS Federal University of Rio de Janeiro.

SO Int Conf AIDS, (1993). Vol. 9, No. 1, pp. 337 (Abstract No. PO-B07-1209).

CY GERMANY: Germany, Federal Republic of

DT Abstract

FS ICA9

LA English

EM 199311

AB CASE REPORT AND RESULTS: Male, 37 y old, homosexual whose CT showed multiple ring-like contrast enhancement hypodense lesions involving deep brain nuclei (Thalamus, basal ganglia). He was given an empirical trial of 30 days with Pyrimethamine and Sulfadiazine with little improvement. Craniotomy was performed and brain biopsy was done. Two cystic lesions have been biopsied, cultured; histopathology and inoculation in guinea pig showed **Mycobacterium tuberculosis** and the other one was positive for *Toxoplasma gondii*. With Rifampin, Isoniazid and Pyrazinamide there was great improvement on the tomographic lesions. CONCLUSION: CNS Tuberculosis appears to be uncommon but should be suspected specially in Brazil, after an empirical trial for Toxoplasmosis has failed to improve clinical status and focal lesions on CT.

CT Check Tags: Animal; Case Report; Human; Male

*Acquired Immunodeficiency Syndrome: CO, complications

Adult

Antitubercular Agents: TU, therapeutic use

*Basal Ganglia Diseases: CO, complications

Basal Ganglia Diseases: MI, microbiology

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Accordingly, these fusion proteins may be used in **treatment** of **HIV-1** or **HIV-2** infection, or may be used as a form of vaccine (no data). Addnl., these chimeric proteins may be used prophylactically in eye drops or in contraceptives (no data). Fusion proteins specific for other viruses can be prepd. by substituting an antibody Fab fragment or viral receptor for the CD4 antigen.

- ST CD4 antigen malaria merozoite protein fusion; receptor virus RBC binding protein fusion; red blood cell binding protein fusion; **HIV** infection **treatment** prevention fusion protein
- IT Proteins, specific or class
RL: BIOL (Biological study)
(EBA-175, fusion products with virus-binding protein of, prophylaxis and treatment of viral infections with)
- IT Immunoglobulins
RL: BIOL (Biological study)
(Fab fragment of anti-viral, fusion products with malaria **parasite** merozoite red blood cell-binding protein, prophylaxis and treatment of viral infections with)
- IT Proteins, specific or class
RL: BIOL (Biological study)
(GBPH (glycophorin binding protein homolog), fusion products with virus-binding protein of, prophylaxis and treatment of viral infections with)
- IT Vaccines
(fusion products of malaria **parasite** merozoite red blood cell-binding protein and CD4 antigen as, prevention of HIV infection with)
- IT Blood transfusion
(fusion products of malaria **parasite** merozoite red blood cell-binding protein and CD4 antigen for prophylaxis in)
- IT Contraceptives
(fusion products of malaria **parasite** merozoite red blood cell-binding protein and CD4 antigen for use in, prevention of HIV infection in relation to)
- IT Protein sequences
(of CD4 antigen-malaria **parasite** merozoite red blood cell-binding protein fusions)
- IT Plasmodium berghei
Plasmodium chabaudi
Plasmodium cynomolgi
Plasmodium gallinaceum
Plasmodium yoelii yoelii
(red blood cell-binding protein of, fusion products with viral receptor, prophylaxis and treatment of viral infections with)
- IT Receptors
RL: BIOL (Biological study)
(viral, fusion products with malaria **parasite** merozoite red blood cell-binding protein, prophylaxis and treatment of viral infections with)
- IT Hepatitis
(B, prophylaxis and treatment of, fusion products of viral receptor and malaria **parasite** merozoite red blood cell-binding protein for)
- IT Hepatitis
(C, prophylaxis and treatment of, fusion products of viral receptor and malaria **parasite** merozoite red blood cell-binding protein for)
- IT Antigens
RL: BIOL (Biological study)
(CD4, fusion products with malaria **parasite** merozoite red blood cell-binding protein of, prophylaxis and treatment of viral infections with)
- IT Hepatitis

- (D, prophylaxis and treatment of, fusion products of viral receptor and malaria **parasite** merozoite red blood cell-binding protein for)
- IT Receptors
RL: BIOL (Biological study)
(Duffy blood-group substances, of **Plasmodium vivax**, fusion products with virus-binding protein of, prophylaxis and treatment of viral infections with)
- IT Blood-group substances
RL: BIOL (Biological study)
(Duffy, receptors, of **Plasmodium vivax**, fusion products with virus-binding protein of, prophylaxis and treatment of viral infections with)
- IT Proteins, specific or class
RL: BIOL (Biological study)
(GBP-130 (glycophorin-binding protein, 130,000-mol.-wt.), fusion products with virus-binding protein of, prophylaxis and treatment of viral infections with)
- IT Proteins, specific or class
RL: BIOL (Biological study)
(P200, fusion products with virus-binding protein of, prophylaxis and treatment of viral infections with)
- IT Antigens
RL: BIOL (Biological study)
(PMMSA (precursor to major merozoite surface antigen), fusion products with virus-binding protein of, prophylaxis and treatment of viral infections with)
- IT Gene
RL: BIOL (Biological study)
(chimeric, for fusion products of malaria **parasite** merozoite red blood cell-binding protein and viral receptor)
- IT Virus, animal
(hepatitis B, receptor for, fusion products with malaria **parasite** merozoite red blood cell-binding protein, prophylaxis and treatment of viral infections with)
- IT Virus, animal
(hepatitis C, receptor for, fusion products with malaria **parasite** merozoite red blood cell-binding protein, prophylaxis and treatment of viral infections with)
- IT Virus, animal
(hepatitis D, receptor for, fusion products with malaria **parasite** merozoite red blood cell-binding protein, prophylaxis and treatment of viral infections with)
- IT Virus, animal
(**human immunodeficiency 1**, infection with, **treatment** of, fusion products of malaria **parasite** merozoite red blood cell-binding protein and CD4 antigen for)
- IT Virus, animal
(**human immunodeficiency 2**, infection with, **treatment** of, fusion products of malaria **parasite** merozoite red blood cell-binding protein and CD4 antigen for)
- IT Microorganism development
(merozoite, malaria **parasite**, blood cell-binding protein of, fusion products with virus-binding protein of, prophylaxis and treatment of viral infections with)
- IT Pharmaceutical dosage forms
(solns., ophthalmic, fusion products of malaria **parasite** merozoite red blood cell-binding protein and CD4 antigen for, prevention of HIV infection with)
- IT 114844-83-6D, Antigen PMMSA (**Plasmodium falciparum** clone gl.1/gl26/pEPG3.3 protein moiety reduced), fusion products with CD4 antigen 151616-85-2 151616-86-3 151616-87-4 151616-88-5D, conjugates with CD4 antigen 151616-89-6 151616-90-9 151616-91-0D, fusion products with P200 or PMMSA of malaria

of Virginia School of Medicine, Charlottesville, VA, United States
 SO ANN. INTERN. MED., (1987) 106/5 (714-718).
 CODEN: AIMEAS
 CY United States
 FS 004 Microbiology
 006 Internal Medicine
 015 Chest Diseases, Thoracic Surgery and Tuberculosis
 030 Pharmacology
 LA English
 AB The widespread emergence of chloroquine-resistant **Plasmodium falciparum** led to the formulation of an effective, fixed combination of two antimalarial agents, pyrimethamine and the long-acting sulfonamide sulfadoxine, for prophylaxis and **treatment**. These drugs act at sequential steps to inhibit the formation of tetrahydrofolate in the **parasite**. Recently, their use for malaria prophylaxis has been associated with severe, at times fatal, cutaneous reactions including erythema multiforme, Stevens-Johnson syndrome, and toxic epidermal necrolysis. These reactions have necessitated a major reassessment of the indications for pyrimethamine-sulfadoxine use and increased the search for pharmacologic, immunologic and behavioral approaches to the prophylaxis and **treatment** of infection with **P. falciparum**. Pyrimethamine-sulfadoxine may be effective in preventing recurrent pneumonia caused by *Pneumocystis carinii* in patients with the acquired **immunodeficiency** syndrome, but life-threatening cutaneous reactions have also been reported in this setting.

CC 037.11.01.03.00. Drug Literature Index/ANTIINFECTIVE AGENTS/Chemotherapeutic agents and antibiotics/Sulfonamides
 037.11.04.00.00. //Antiprotozoal drugs
 038.29.00.00.00. Adverse Reactions Titles/ANTIPROTOZOAL DRUGS

CT EMTAGS: **priority journal** (0007); skin, hair, nails and sweat glands (0980); intoxication (0302); blood and hemopoietic system (0927); immunological factors (0136); therapy (0160); adverse drug reaction (0198); oral drug administration (0181); review (0001); **human** (0888); infection (0310); protozoon (0751); bacterium (0762)

Medical Descriptors:
 *fansidar
 ***plasmodium falciparum**
 *pneumocystis carinii
 *pyrimethamine
 *sulfadoxine
 *erythema multiforme
 *stevens johnson syndrome
 *toxic epidermal necrolysis
 *megaloblastic anemia
 *nephrotoxicity
 *liver toxicity
 *drug hypersensitivity
 chloroquine
 *drug mixture
 *pharmacotherapy
 *drug efficacy
 *adverse drug reaction
 *skin toxicity
 ***acquired immune deficiency syndrome**
 *prophylaxis
 drug resistance

Drug Descriptors:
 quinine
 amodiaquine
 proguanil
 mefloquine

tetracycline derivative
diethyltoluamide
primaquine

CN Fansidar; Camoquin; Flavoquine; Paludrine
CO Hoffmann la roche (United States); Ici (United Kingdom); Parke davis
(United Kingdom); Roussel (France)

L94 ANSWER 84 OF 108 HCAPLUS COPYRIGHT 1998 ACS DUPLICATE 10

AN 1988:147004 HCAPLUS

DN 108:147004

TI Effect of benzalkonium chloride on HIV and related infections and on
other infectious agents

AU Wainberg, M. A.; Bleau, G.

CS Lady Davis Inst. Med. Res., Sir Mortimer B. Davis - Jewis Gen.
Hosp., Montreal, PQ, Can.

SO Arch. AIDS Res. (1987), 1(1), 57-68

CODEN: AARSE9

DT Journal

LA English

CC 10-5 (Microbial Biochemistry)

Section cross-reference(s): 1

AB Benzalkonium chloride can be used to greatly reduce HIV-1 (
human immunodeficiency virus) reverse transcriptase activity
upon exposure to virus. Such inactivation takes place in a
concn.-dependent manner. Furthermore, this drug is able at concns.
of 0.05% and higher, in aq. soln., to completely destroy HIV-1
infectivity, when tested under these conditions. Exposure of free
virus to the interior of a benzalkonium-contg. condom appeared to
greatly reduce potential infectivity. Similar results were obtained
when HIV-1-infected H-9 cells were exposed to benzalkonium within
the interior of a condom, prior to exposure to target cells.
Neither of two latex rubber condoms tested were permeable to HIV-1
or the HIV-1-infected cells. Following puncture of the condom wall
by a 18-gauge needle and the recovery and testing of the contents of
the condom from the outside, it was found that no free HIV-1
survived exposure to the interior of a benzalkonium-contg. device,
whereas some HIV-1 did survive exposure to the interior of a
non-drug-contg. condom. However, some residual infectivity could be
detected on the part of HIV-1-infected H-9 cells which had been
exposed to the interior of a benzalkonium-contg. condom.
Benzalkonium chloride, at moderate concns., was viricidal for herpes
simplex virus type 2 and cytomegalovirus. However, this drug had no
effect on reactivity of hepatitis B surface antigen with specific
antibody. A transient bacteriostatic effect was obsd. with regard
to exposure of benzalkonium chloride to **Mycobacterium**
tuberculosis.

ST benzalkonium chloride **inhibition human**
immunodeficiency virus; virucide benzalkonium chloride; AIDS
virus benzalkonium chloride

IT Virucides and Virustats
(benzalkonium chloride)

IT **Mycobacterium tuberculosis**
(inhibition of, by benzalkonium chloride)

IT Quaternary ammonium compounds, biological studies
RL: BIOL (Biological study)

(alkylbenzyltrimethyl, chlorides, **human**
immunodeficiency virus **inhibition** by)

IT Virus, animal
(cytomegalo-, inhibition of, by benzalkonium chloride)

IT Virus, animal
(herpes simplex 2, inhibition of, by benzalkonium chloride)

IT Virus, animal
(**human immunodeficiency**, **inhibition**
of, by benzalkonium chloride)

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IT 9068-38-6, Reverse transcriptase
RL: PROC (Process)
(of **human immunodeficiency virus**,
benzalkonium chloride **inhibition** of)

L94 ANSWER 85 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1987:512471 HCAPLUS
DN 107:112471
TI Activity of ciprofloxacin and other fluorinated quinolones against
mycobacteria
AU Young, Lowell S.; Berlin, O. George W.; Inderlied, Clark B.
CS Kuzell Inst. Arthritis Infect. Dis., San Francisco, CA, 94115, USA
SO Am. J. Med. (1987), 82(4A), 23-6
CODEN: AJMEAZ; ISSN: 0002-9343
DT Journal
LA English
CC 10-5 (Microbial Biochemistry)
AB The new fluorinated quinolones display interesting but variable
activity against mycobacteria. Almost all compds. tested
(ciprofloxacin, ofloxacin, enoxacin, norfloxacin, difloxacin, I-934,
A-56620, and megalone) inhibit **Mycobacterium**
tuberculosis at achievable serum concns., with ciprofloxacin
and ofloxacin most active by wt. (minimal inhibitory concn. at which
growth of 90% of strains is inhibited is .ltoreq.1 .mu.g/mL). The
growth of *M. kansasii*, *M. xenopi*, and *M. fortuitum* is also well
inhibited by these agents in the same range of concns. Activity
against the *M. avium* complex is method-dependent, with growth of
perhaps one-third of the strains isolated from patients with the
acquired **immune deficiency syndrome**
inhibited by ciprofloxacin. Detn. of individual drug
efficacy data in exptl. mycobacterial infections is not a practical
goal. However, combination therapy studies are in progress using
murine models of both **M. tuberculosis** and *M.*
avium challenges. Ofloxacin has been used with some success in
human patients with pulmonary tuberculosis. Oral
administration may be an important advantage, and, when used in
combination with other active agents, the new quinolones may have a
useful role in treating mycobacterial infections.
ST mycobacteria fluorinated quinolone ciprofloxacin; tuberculostatic
ciprofloxacin ofloxacin enoxacin norfloxacin megalone
IT *Mycobacterium avium*
Mycobacterium fortuitum
Mycobacterium kansasii
Mycobacterium tuberculosis
Mycobacterium xenopi
(fluorinated quinolone sensitivity of)
IT Tuberculostatics
(fluorinated quinolones)
IT 70458-96-7, Norfloxacin 74011-58-8, Enoxacin 82419-36-1,
Ofloxacin 85721-33-1, Ciprofloxacin 91188-00-0, CI-934
98105-99-8, A-56620 98106-17-3, Difloxacin 110158-59-3
RL: BAC (Biological activity or effector, except adverse); BIOL
(Biological study)
(*Mycobacterium* sensitivity to)

L94 ANSWER 86 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1986:502597 HCAPLUS
DN 105:102597
TI Silver sulfonamide-complexes of diamines as antimicrobial agents
IN Scovill, John P.; Filippen-Anderson, Judith L.; Gilardi, Richard;
Miller, Robert E.; Milhous, Wilber K.
PA USA
SO U. S. Pat. Appl., 36 pp. Avail NTIS Order No. PAT-APPL-6-771 981.
CODEN: XAXXAV

PI US 771981 A0 860328
AI US 85-771981 850903
DT Patent
LA English
CC 63-6 (Pharmaceuticals)
AB Antimicrobial (esp. bacteria and protozoa) agents comprise Ag-sulfonamide complexes of aliph. or arom. diamines having Cl-3 in the moiety bridging the 2 amino groups. Thus, the Ag metachloridine complex with 1,2-diaminoethane was prepd. by mixing a water soln. contg. 2.84 g metachloridine and 5 mL 1,2-diaminoethane with a soln. contg. 1.7 g AgNO3 and 3 mL 1,2-diaminoethane and allowing to stand for 3 h. The yield was 75% and the complex melted at 168-169.degree..
ST antimicrobial silver sulfonamide diamine complex; protozoacide silver sulfonamide diamine complex; bactericide silver sulfonamide diamine complex
IT **Escherichia coli**
Klebsiella pneumoniae
Proteus mirabilis
Pseudomonas aeruginosa
Shigella dysenteriae
Staphylococcus aureus
Streptococcus faecalis
(inhibition of, with silver-metachloridine-aminoethylpyridine complex)
IT Plasmodium **falciparum**
Trypanosoma rhodesiense
(inhibition of, with silver-sulfonamide-diamine complexes)
IT Antimalarials
Bactericides, Disinfectants, and Antiseptics
Protozoacides
Trypanosomicides
(silver-sulfonamide-diamine complexes)
IT 103937-71-9P 103937-72-0P 103937-73-1P 103937-74-2P
RL: PREP (Preparation)
(prepn. of, as antimicrobial agent)
IT 22199-08-2
RL: RCT (Reactant)
(reaction of, with aminomethylpyridine)
IT 563-63-3
RL: RCT (Reactant)
(reaction of, with metachloridine and aminomethylpyridine)
IT 7761-88-8, reactions
RL: RCT (Reactant)
(reaction of, with metachloridine and diamines)
IT 3731-51-9
RL: RCT (Reactant)
(reaction of, with silver acetate and metachloridine)
IT 565-36-6
RL: RCT (Reactant)
(reaction of, with silver compds. and diamines)
IT 107-15-3, reactions 109-76-2
RL: RCT (Reactant)
(reaction of, with silver nitrate and metachloridine)
L94 ANSWER 87 OF 108 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 86-225208 [34] WPIDS
CR 85-263120 [42]
DNC C86-097206
TI Compsn. of microbially produced recombinant IL-2 - used for treatment of immuno modulatory indications.
DC B04 C03
IN FERNANDES, P M; TAFORO, T A
PA (CETU) CETUS CORP

CYC 1
PI US 4604377 A 860805 (8634)* 8 pp
ADT US 4604377 A US 85-715152 850321
PRAI US 84-594350 840328; US 85-715152 850321
IC A61K037-02; A61K039-39; A61K045-02; C07K013-00
AB US 4604377 A UPAB: 941122

Recombinant IL-2 compsn. (I) comprises a sterile lyophilised mixt. of (i) a selectively oxidised microbially produced recombinant IL-2, which is free of non-IL-2 protein and is at least 95% pure recombinant IL-2, and contains less than 5 ng endotoxin per 100,000 units of IL-2 activity; (ii) a water soluble carrier which does not affect the stability of (i); and (iii) a surface active agent to ensure the water solubility of (i).

For therapy (I) is dissolved in an aq. parenteral injection, the soln. contg. 0.01-2 mg(i), (also claimed).

USE - (I) is useful for **treatment** of **immunodeficiency** states, acquired, inborn or induced by chemotherapy, immunotheapy or irradiation, enhancement of cell-mediated immune responses in the therapy of viral, **parasitic**, bacterial, malignant, fungal, prozoal or mycobacterial or other infectious diseases; induction of enhanced immunologic response of cells ex vivo in the treatment of infectious, malignant, rhumatic or autoimmune diseases; treatment of rhumatism of other inflammatory arthidites; treatment of diseases of abnormal immune response by multiple sclerosis, systemic lupus erythematosus, glomerulonephritis or hepatitis; regulation of haematopoietic tumours or pre-malignant or aplastic abnormalities of haematopoietic tissue; as an adjuvant in induction of cell-mediated or humoral response to vaccines or antigens; as a mediator or modified of CNS function; for treatment of malignant or pre-malignant diseases in combination with othef therapies; for treatment of **m.tuberculosis** in combination with drug therapy; and for prophylaxis against infectious diseases.

Dwg.0/1

Dwg.0/1

FS CPI

FA AB

MC CPI: B04-C01; B10-A09A; B12-A01; B12-A02C; B12-A04; B12-A06;
B12-B01; B12-B04; B12-C10; B12-D02A; B12-D03; B12-D07; B12-D09;
B12-E02; B12-G02; B12-G03; B12-G07; B12-M09; C04-C01; C10-A09A;
C12-A01; C12-A02C; C12-A04; C12-A06; C12-B01; C12-B04; C12-C10;
C12-D02A; C12-D03; C12-D07; C12-D09; C12-E02; C12-G02; C12-G03;
C12-G07; C12-M09

L94 ANSWER 88 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1986:545665 HCAPLUS

DN 105:145665

TI 5-(N-Arylnortropan-3-yl)- and 5-(N-arylpiperidin-4-yl)-2,4-diaminopyrimidines. Novel inhibitors of dihydrofolate reductase

AU Maag, Hans; Locher, Rita; Daly, John J.; Kompis, Ivan

CS F. Hoffmann-La Roche und Co., Ltd., Basel, CH-4002, Switz.

SO Helv. Chim. Acta (1986), 69(4), 887-97

CODEN: HCACAV; ISSN: 0018-019X

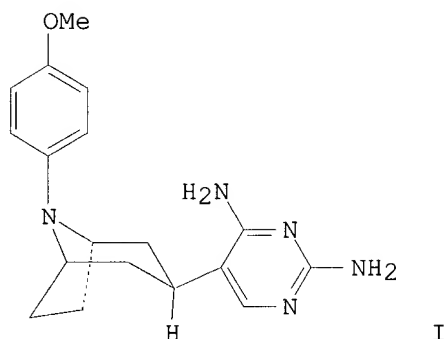
DT Journal

LA English

CC 1-3 (Pharmacology)

Section cross-reference(s): 7, 10, 28

GI



- AB Based on a computer-assisted anal. of the 3-dimensional structure of the binary complex of *Escherichia coli* dihydrofolate reductase (DHFR) with methotrexate, 5-(N-arylnortropan-3-yl)- and 5-(N-arylpiperidin-4-yl)-2,4-diaminopyrimidines were designed as inhibitors of DHFR. Synthesis of the designed compds. have been carried out. The most potent compd. I [94635-30-0] inhibited *E. coli* DHFR with $K_i = 0.49$.times. $-9M$. The activities within the series of compds. synthesized could be rationalized by mol.-modeling expts. Several compds. within the presented series exhibit antimalarial activities in vitro and in vivo.
- ST aminopyrimidine prepn dihydrofolate reductase inhibitor structure; antimalarial aminopyrimidine
- IT Antimalarials
((arylnortropanyl)- and (arylpiperidinyl)diaminopyrimidines)
- IT Crystal structure
(diamino[(methoxyphenyl)azabicyclooctyl]pyrimidines)
- IT *Plasmodium falciparum*
(diaminopyrimidines activity against)
- IT *Escherichia coli*
Lactobacillus casei
Liver, composition
(dihydrofolate reductase from, diaminopyrimidines inhibition of)
- IT Molecular structure-biological activity relationship
(tetrahydrofolate dehydrogenase-inhibiting, of (arylnortropanyl)- and (arylpiperidinyl)diaminopyrimidines)
- IT 105-56-6
RL: RCT (Reactant)
(Knoevenagel condensation of, with (dimethoxyphenyl)azabicyclooct anone)
- IT 33205-16-2
RL: RCT (Reactant)
(Knoevenagel condensation of, with Et cyanoacetate)
- IT 10272-07-8
RL: RCT (Reactant)
(Mannich reaction of, with oxoglutaric acid and dimethoxytetrahydrofuran)
- IT 50-01-1
RL: BIOL (Biological study)
(condensation of, with Et cyano(dimethoxyphenyl)azabicyclooctane xacetate)
- IT 9002-03-3
RL: BIOL (Biological study)
(inhibitors of, diaminopyrimidines as)
- IT 56525-68-9P 104383-34-8P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and Dieckmann condensation and decarboxylation of)

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IT 35193-97-6P 94634-89-6P 94635-24-2P
 RL: SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and Knoevenagel condensation with Et cyanoacetate)

IT 94634-90-9P 94635-25-3P
 RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and catalytic hydrogenation of)

IT 94634-92-1P 94635-17-3P 94635-21-9P 94635-27-5P 104404-87-7P
 104404-88-8P
 RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and chlorination of)

IT 94634-91-0P 94635-26-4P 104404-85-5P 104404-86-6P
 RL: SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and condensation with guanidine HCl)

IT 94635-30-0P
 RL: SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and dehydrofoalte reductase inhibiting and antimalarial
 activity of, structure in relation to)

IT 156-81-0DP, derivs. 94635-31-1P 94635-32-2P 94635-33-3P
 104383-32-6P 104383-33-7P
 RL: SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and dihydrofolate reductase-inhibiting and antimalarial
 activities of, structure in relation to)

IT 94634-88-5P 94635-23-1P
 RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and hydrolysis of)

IT 94635-14-0P 94635-15-1P 94635-18-4P 94635-19-5P 94635-22-0P
 94635-28-6P 104404-89-9P 104404-90-2P
 RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
 (prepn. and redn. of)

IT 104-94-9
 RL: RCT (Reactant)
 (reaction of, with Et acrylate)

IT 542-05-2
 RL: RCT (Reactant)
 (reaction of, with dimethoxyaniline and dimethoxytetrahydrofuran)

IT 140-88-5
 RL: RCT (Reactant)
 (reaction of, with methoxyaniline)

IT 696-59-3
 RL: RCT (Reactant)
 (reaction of, with oxoglutaric acid and dimethoxyaniline)

L94 ANSWER 89 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 86110803 EMBASE
 TI [Antiparasitic drug therapy adapted to particular endemic regions].
 INDICATIONS PARTICULIERES DE CERTAINS TRAITEMENTS ANTIPARASITAIRES
 EN ZONES D'ENDEMIE.
 AU Gendrel D.; Nardou M.; Richard-Lenoble D.; Kombila M.
 CS Centre Universitaire des Sciences de la Sante, BP 4009, Libreville,
 Gabon
 SO ARCH. FR. PEDIATR., (1985) 42/SUPPL. 2 (983-985).
 CODEN: AFPEAM
 CY France
 LA French
 SL English
 AB In endemic regions, certain anti-**parasitic** therapies are
 automatically prescribed when confronted with apparently benign
 childhood disorders. The diagnostic differentiation between a simple
 febrile seizure provoked by **Plasmodium falciparum**
 is often impossible, requiring the initial use of intravenous
 quinine. Helminth or Giardia infestations often aggravate the
 chronic diarrhea of malnutrition, or are revealed with
 corticosteroid therapy, necessitating the initiation of appropriate
treatment. In addition, the frequent association of

typhoid and schistosomiasis, requires therapy for both in order to prevent relapses.

CC 004.10.01.05.00.
 004.10.05.01.00.
 004.10.06.03.00.
 004.10.08.01.00.
 007.07.03.00.00.
 007.12.06.00.00.
 007.30.05.00.00.
 007.36.01.01.00.
 017.03.07.00.00.
 017.03.08.00.00.
 030.20.08.00.00.
 030.20.08.04.00.
 030.20.09.00.00.
 037.11.03.00.00. Drug Literature Index/ANTIINFECTIVE AGENTS/Anthelmintics
 037.11.04.00.00. //Antiprotozoal drugs

CT EMTAGS: **priority journal** (0007); therapy (0160); oral drug administration (0181); review (0001); epidemiology (0400); geographical aspects (0401); infection (0310); prevention (0165); **human** (0888); nematode (0754); microorganism (0724)
 Medical Descriptors:
 *pharmacotherapy
 *giardia
 *parasitosis
 *plasmodium falciparum
 *typhoid fever
 *schistosomiasis
 *mebendazole
 *albendazole
 *chloroquine
 *clioquinol
 *quinine formate
 *tiabendazole
 *metronidazole
 *niridazole
 quinine
 diarrhea
 malnutrition
 tropic medicine

CN Quinoform

L94 ANSWER 90 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS
 AN 85:143013 BIOSIS
 DN BR29:33009
 TI IN-VITRO SUSCEPTIBILITY OF MYCOBACTERIA TO ANSAMYCIN.
 AU HEIFETS L; LINDHOLM-LEVY P; ISEMAN M
 CS NATL. JEWISH HOSP./RES. CENT., DENVER, COLO.
 SO 85TH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR MICROBIOLOGY, LAS VEGAS, NEV., USA, MAR. 3-7, 1985. ABSTR ANNU MEET AM SOC MICROBIOL 85 (0). 1985. 107. CODEN: ASMACK ISSN: 0094-8519
 DT Conference
 LA English
 ST ABSTRACT MYCOBACTERIUM-AVIUM MYCOBACTERIUM-INTRACELLULARE
MYCOBACTERIUM-TUBERCULOSIS HUMAN RIFAMPIN
 ANTIBACTERIAL-DRUG BACTERICIDAL BACTERIOSTATIC ACQUIRED
IMMUNE DEFICIENCY SYNDROME BACTEC RADIOMETRIC
 SYSTEM MINIMUM **INHIBITORY** CONCENTRATION

RN 13292-46-1 (RIFAMPIN)
 51374-14-2 (ANSAMYCIN)

CC General Biology-Symposia, Transactions and Proceedings of
 Conferences, Congresses, Review Annuals 00520
 Radiation-Radiation and Isotope Techniques 06504
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- Biochemical Studies-General 10060
 Pathology, General and Miscellaneous-Necrosis 12510
 Pathology, General and Miscellaneous-Therapy 12512
 Pharmacology-Clinical Pharmacology *22005
 Physiology and Biochemistry of Bacteria 31000
 Microbiological Apparatus, Methods and Media 32000
 Immunology and Immunochemistry-Bacterial, Viral and Fungal 34504
 Immunology and Immunochemistry-Immunopathology, Tissue Immunology
 *34508
 Medical and Clinical Microbiology-General; Methods and Techniques
 36001
 Medical and Clinical Microbiology-Bacteriology *36002
 Medical and Clinical Microbiology-Virology *36006
 Chemotherapy-Antibacterial Agents *38504
 BC Retroviridae-Oncovirinae 02244
 Mycobacteriaceae 05822
Hominidae 86215
- L94 ANSWER 91 OF 108 HCAPLUS COPYRIGHT 1998 ACS
 AN 1986:223818 HCAPLUS
 DN 104:223818
 TI Effect of heat on specific proteins in **human** milk
 AU Lyster, Richard L. J.; Hunjan, Manjit; Hall, Eveline D.
 CS Natl. Inst. Res. Dairy., Shinfield/Reading, RG2 9AT, UK
 SO Nestle Nutr. Workshop Ser. (1984), 5(Hum. Milk Banking), 93-100
 CODEN: NNWSDT; ISSN: 0742-2806
 DT Journal
 LA English
 CC 17-8 (Food and Feed Chemistry)
 AB **Human** milk samples heated at 62.5.degree. for 30 min
 reduced Escherichia coli counts to acceptable levels, denatured alk.
 phosphatase [9001-78-9] so that it remained a useful test for
 proper pasteurization (**Mycobacterium tuberculosis**
 is less heat-stable than is the enzyme), but partly degraded
 lactoferrin and serum IgA. Heating for 30 min at 57.degree. showed
 no loss of IgA on lactoferrin, adequate redn. of the E. coli count,
 but did not inactivate and thus minimized the usefulness of using
 alk. phosphatase as a test enzyme for proper pasteurization; lipase
 [9001-62-1] may be substituted as a test enzyme at this temp.
 ST milk **human** pasteurization protein denaturation
 IT **Escherichia coli**
 (growth **inhibition** of, of **human** milk,
 pasteurization method in relation to)
 IT Enzymes
 Lactoferrins
 RL: PROC (Process)
 (of **human** milk, heat denaturation of)
 IT Immunoglobulins
 RL: PROC (Process)
 (A, of **human** milk, heat denaturation of)
 IT Milk
 (**human**, proteins of, heat denaturation of)
 IT 9001-62-1 9001-78-9
 RL: PROC (Process)
 (of **human** milk, denaturation of, as index of proper
 pasteurization)
- L94 ANSWER 92 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS
 AN 84:256581 BIOSIS
 DN BA77:89565
 TI ANTI BACTERIAL ACTIVITY OF PALMITOYL TUBERACTINAMINE N AND DI-BETA
 LYSYL CAPREOMYCIN IIA.
 AU YAMADA T; YAMANOUCI T; ONO Y; NAGATA A; WAKAMIYA T; TESHIMA T; SHIBA
 T

- CS RES. INST. FOR MICROBIAL DISEASES, OSAKA UNIV., 3-1 YAMADA-OKA,
SUITA, OSAKA 565, JPN.
- SO J ANTIBIOT (TOKYO) 36 (12). 1983 (RECD. 1984). 1729-1734. CODEN:
JANTAJ ISSN: 0021-8820
- LA English
- AB Palmitoyltuberactinamine N (Pal-Tua N) and di-.beta.-lysylcapreomycin
IIA (di-.beta.-Lys-Cpm IIA), synthetic derivatives of the
antituberculous agent tuberactinomycin (Tum) and capreomycin (Cpm),
respectively, were tested for antibacterial activity. Pal-Tua N
inhibited tuberactinomycin-resistant Mycobacterium smegmatis,
Escherichia coli, Corynebacterium diphtheriae,
Staphylococcus aureus and Streptococcus pyogenes, and had no activity
against **M. tuberculosis**. Di-.beta.-Lys-Cpm IIA
inhibited the growth of laboratory-derived Tum-resistant M.
smegmatis and **M. tuberculosis** as well as
Tum-resistant **M. tuberculosis** from patients, with
1 exceptional case.
- ST MYCOBACTERIUM-SMEGMATIS MYCOBACTERIUM-TUBERCULOSIS
ESCHERICHIA-COLI CORYNEBACTERIUM-DIPHThERIAE STAPHYLOCOCCUS-AUREUS
STREPTOCOCCUS-PYOGENES HUMAN TUBER ACTINOMYCIN CAPREOMYCIN
ANTIBACTERIAL-DRUG
- RN 11003-38-6 (CAPREOMYCIN)
11075-36-8 (TUBER ACTINOMYCIN)
- CC Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous-Therapy 12512
Pharmacology-General *22002
Physiology and Biochemistry of Bacteria 31000
Medical and Clinical Microbiology-Bacteriology *36002
Chemotherapy-Antibacterial Agents *38504
- BC Enterobacteriaceae 04810
Micrococcaceae 05510
Streptococcaceae 05514
Coryneform Group of Bacteria 05814
Mycobacteriaceae 05822
Hominidae 86215
- L94 ANSWER 93 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 11
- AN 81:247662 BIOSIS
- DN BA72:32646
- TI **MALARIO THERAPY AND CANCER.** X
- AU GREENTREE L B
- CS 3111 EAST BROAD ST., COLUMBUS, OHIO.
- SO MED HYPOTHESES 7 (1). 1981. 43-50. CODEN: MEHYDY ISSN: 0306-9877
- LA English
- AB **Malariotherapy** [using the Madagascar strain of Plasmodium
vivax] merits a clinical trial as an adjuvant to conventional cancer
therapy. This particular modality of treatment is a most potent
stimulus of macrophage activity. These scavenger cells are widely
believed to be an essential arm in the host's immune defenses against
malignant disease, both as regards the processing of antigens and as
killers of tumor cells. **Malariotherapy** was used to
effectively treat some 16,000 patients with paretic neurosyphilis in
1 institution alone, before the advent of the penicillin age, and has
proved to be a particularly safe modality of treatment.
- ST HUMAN PLASMODIUM-VIVAX MADAGASCAR STRAIN MACROPHAGE IMMUNE DEFENSE
PENICILLIN ANTIINFECTIVE NEURO SYPHILIS THERAPY SAFETY
- RN 1406-05-9 (PENICILLIN)
- CC Cytology and Cytochemistry-Animal 02506
Biochemical Studies-General 10060
Pathology, General and Miscellaneous-Therapy 12512
Metabolism-General Metabolism; Metabolic Pathways 13002
Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies 15004
Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and
Reticuloendothelial System *15008

Nervous System-General; Methods 20501
 Nervous System-Pathology *20506
 Pharmacology-Immunological Processes and Allergy *22018
 Neoplasms and Neoplastic Agents-Therapeutic Agents; Therapy *24008
 Immunology and Immunochemistry-General; Methods *34502
 Immunology and Immunochemistry-Immunopathology, Tissue Immunology
 *34508
 Immunology, Parasitological *35000
 Chemotherapy-General; Methods; Metabolism *38502
 Food and Industrial Microbiology-Food and Beverage Spoilage and
 Contamination *39002
 Parasitology-Medical *60504
 Invertebrata, Comparative and Experimental Morphology, Physiology and
 Pathology-Protozoa 64002
 BC Spirochaetaceae 04510
 Sporozoa 35400
 Hominidae 86215

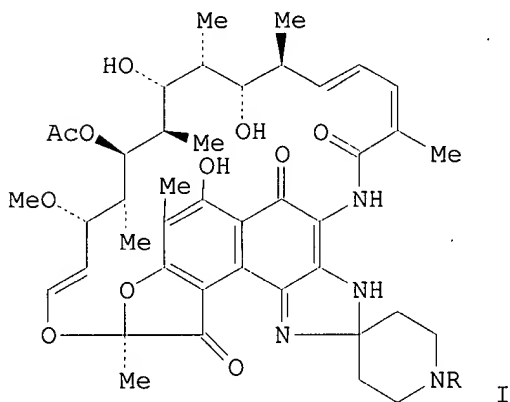
L94 ANSWER 94 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS
 AN 83:192595 BIOSIS
 DN BA75:42595
 TI INVESTIGATIONS ON THE BACTERIAL INTESTINAL FLORA IN CHILDREN INVADED
 WITH **ASCARIS-LUMBRICOIDES**.
 AU ZAN T K; ESEVA Z
 CS SCI. RES. INST. INFECT. PARASIT. DIS., SOFIA, BULG.
 SO KHELMINTOLOGIYA 12 (0). 1981 (RECD. 1982). 31-35. CODEN: KHELDD
 ISSN: 0324-1947
 LA Bulgarian
 AB There were 108 children aged 7-10 yr from a village in Southwestern
 Bulgaria investigated. Quantitative and qualitative investigations of
 the intestinal microflora as well as **parasitic**
 investigations of fecal samples before and a mo. after treatment with
 Decaris were carried out. The intensity of the invasion with *A.*
lumbricoides among the investigated children was comparatively high
 (51.8%). Decaris is one of the medicines with a good curative effect
 (96%). No difference in the microbial number of the aerobic
 intestinal flora in children with ascaridiasis before and after
 treatment was established. The quantity of the anaerobic
 bifidobacteria in children with ascaridiasis was greater than in
 those without ascaridiasis. The difference was statistically
 significant. The number of the isolated enteropathogenic
Escherichia coli in children with ascaridiasis
 before **treatment** was greater than in those without
 ascaridiasis. The difference was statistically significant. A
 decrease was observed in the number of the isolated **E.**
coli after **treatment** of children with ascaridiasis.
 A difference in the quantity of the isolated enteropathogenic *E. coli*
 was not observed in children without ascaridiasis either before or
 after treatment. Thus, the treatment of the ascaridiasis probably
 should precede that of the intestinal infections in cases when
 combinations of *A. lumbricoides* and pathogenic intestinal bacteria
 occur.

ST BIFIDOBACTERIA ESCHERICHIA-COLI DECARIS ANTIPARASITIC-DRUG
 SOUTHWESTERN BULGARIA
 RN 16595-80-5 (DECARIS)
 CC Mathematical Biology and Statistical Methods 04500
 Social Biology; Human Ecology 05500
 Biochemistry-Gases 10012
 Pathology, General and Miscellaneous-Comparative 12503
 Pathology, General and Miscellaneous-Therapy *12512
 Digestive System-General; Methods 14001
 Digestive System-Physiology and Biochemistry *14004
 Digestive System-Pathology *14006
 Pharmacology-Clinical Pharmacology 22005

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Pharmacology-Digestive System *22014
 Pediatrics *25000
 Physiology and Biochemistry of Bacteria 31000
 Medical and Clinical Microbiology-General; Methods and Techniques
 36001
 Medical and Clinical Microbiology-Bacteriology *36002
 Chemotherapy-Antiparasitic Agents *38510
 Parasitology-Medical *60504
 Invertebrata, Comparative and Experimental Morphology, Physiology and
 Pathology-Aschelminthes 64016
 BC Bacteria-Unspecified 04000
 Enterobacteriaceae 04810
 Actinomycetaceae 05810
 Nematoda 51300
Hominidae 86215

L94 ANSWER 95 OF 108 HCAPLUS COPYRIGHT 1998 ACS
 AN 1981:10879 HCAPLUS
 DN 94:10879
 TI Biological activity of a new class of rifamycins
 spiropiperidylrifamycins
 AU Sanfilippo, A.; Della Bruna, C.; Marsili, L.; Morvillo, E.;
 Pasqualucci, C. R.; Schioppacassi, G.; Ungheri, D.
 CS Res. Lab., Farmitalia Carlo Erba, Milan, Italy
 SO J. Antibiot. (1980), 33(10), 1193-8
 CODEN: JANTAJ; ISSN: 0021-8820
 DT Journal
 LA English
 CC 1-3 (Pharmacodynamics)
 GI



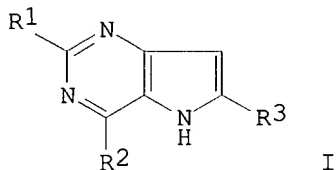
AB The biol. properties of spiro-piperidyl-rifamycins (I), a new class of rifamycin antibiotics, are described. In these derivs. the positions 3 and 4 have been incorporated into an imidazolyl ring bearing a spiro-piperidyl group N substituted with linear and branched aliph. chains. The in vitro antibacterial activity against *Staphylococcus aureus* and *Mycobacterium tuberculosis* increases with the no. of the carbon atoms in the linear side chain, whereas the inhibitory effect on *Escherichia coli* is lowered. The antibacterial activity is only marginally affected by branching of the side chain. In vivo (exptl. infections of mice), the optimal therapeutic activity against *M. tuberculosis* is shown by compds. bearing 3-5 carbon atoms as a linear or branched side chain; in comparison with rifampicin, the potency of these derivs. is 2-3 times higher. The finding is in a good agreement

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with the exceptional tissue tropism, which seems to be a favorable property of this group of derivs.

ST spiropiperidyl rifamycin deriv antibiotic structure; structure
activity spiropiperidyl rifamycin deriv
IT Antibiotics
(spiropiperidyl rifamycins as, structure in relation to)
IT Molecular structure-biological activity relationship
(antibiotic, of spiropiperidyl rifamycins)
IT 6998-60-3D, spiropiperidyl derivs. 62295-71-0 71072-23-6
71072-29-2 72544-08-2 72544-09-3 72544-14-0 72544-15-1
72559-05-8 72559-06-9 72559-07-0 75903-10-5 75903-11-6
75903-12-7 75903-13-8
RL: BAC (Biological activity or effector, except adverse); BIOL
(Biological study)
(antibiotic activity of, structure in relation to)

L94 ANSWER 96 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1980:69323 HCAPLUS
DN 92:69323
TI Pyrrolo[3,2-d]pyrimidines as potential antitumor agents
AU Kravchenko, A. I.; Chernov, V. A.; Shcherbakova, L. I.; Filitis, L.
N.; Pershin, G. N.; Sokolova, V. N.
CS Vses. Nauchno-Issled. Khim.-Farm. Inst., Moscow, USSR
SO Farmakol. Toksikol. (Moscow) (1979), 42(6), 659-65
CODEN: FATOAO; ISSN: 0014-8318
DT Journal
LA Russian
CC 1-3 (Pharmacodynamics)
Section cross-reference(s): 3
GI



AB Only 1 of the 44 pyrrolopyrimidines I tested, 2,6-dimethyl-4-sulfanilamidopyrrolo(3,2-d)pyrimidine [72549-78-1], showed marked **inhibitory** activity against **Escherichia coli** in vitro, having a minimal **inhibitory** concn. (MIC) of 1 .mu.g/mL. Eight of the compds. had MIC values .ltoreq.1 .mu.g/mL against Lactobacillus casei and 11 had similar MICs against **Mycobacterium tuberculosis** H37Rv. In addn. to showing high antibacterial activity, 6-methyl-4-mercapto-2-phenylpyrrolo[3,2-d]pyrimidine [72168-74-2] also had marked antitumor activity against sarcoma 180 in **mice** and increased the life span of animals with leukemia L-1210.
ST pyrrolopyrimidine deriv bactericide antitumor
IT Bactericides, Disinfectants and Antiseptics
Neoplasm inhibitors
(pyrrolopyrimidines)
IT Molecular structure-biological activity relationship
(bactericidal, of pyrrolopyrimidines)
IT Molecular structure-biological activity relationship
(neoplasm-inhibiting, of pyrrolopyrimidines)
IT 272-50-4D, derivs. 41040-25-9 41040-27-1 41040-28-2
41040-29-3 41040-39-5 52617-58-0 52617-59-1 52617-60-4
52617-61-5 52617-62-6 52617-69-3 52617-72-8 52659-60-6
52739-36-3 72168-68-4 72168-69-5 72168-70-8 72168-71-9

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72168-72-0 72168-73-1 72168-74-2 72549-60-1 72549-61-2
 72549-62-3 72549-63-4 72549-64-5 72549-65-6 72549-66-7
 72549-67-8 72549-68-9 72549-69-0 72549-70-3 72549-71-4
 72549-72-5 72549-73-6 72549-74-7 72549-75-8 72549-76-9
 72549-77-0 72549-78-1 72549-79-2 72549-80-5 72549-81-6
 72561-16-1

RL: BIOL (Biological study)

(bactericidal and neoplasm-inhibiting activity of, structure in relation to)

L94 ANSWER 97 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1979:180801 HCAPLUS

DN 90:180801

TI Cefazedone: microbiological evaluation in comparison with cephalothin and cefazolin

AU Wahlig, H.; Dingeldein, E.; Mitsuhashi, S.; Kawabe, H.

CS Dep. Chemother., E. Merck, Darmstadt, Ger.

SO Arzneim.-Forsch. (1979), 29(2A), 369-78

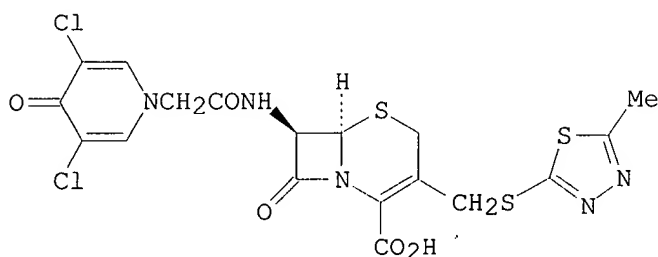
CODEN: ARZNAD; ISSN: 0004-4172

DT Journal

LA English

CC 3-2 (Biochemical Interactions)

GI



AB In low concns., cefazedone Na (I Na) [63521-15-3] was active against a large no. of gram-pos. and gram-neg. organisms susceptible to other .beta.-lactam antibiotics. I was several times more potent than cefazolin Na [27164-46-1] and cephalothin Na [58-71-9] against Staphylococcus aureus and even more so against Streptococcus pyogenes. Also enterococci (Streptococcus faecalis), which are usually resistant to cephalosporins, were inhibited by 90% by I. The min. inhibitory concns. of I against gram-neg. pathogens were comparable to those of cefazolin. Proteus mirabilis strains were inhibited by only 70%. I acted bactericidally in low concns. with only small differences between the min. inhibitory and the min. bactericidal levels. The effects of inoculum size, pH, **human** serum, and different culture media on the I antibacterial activity were negligible. Max. activity was obsd. at pH 6.0. Stability in body fluids and buffer solns. were investigated at various temps. I could be stored for .gtoreq.8 wk without loss of activity at -30.degree. in **human** serum and urine as well as in phosphate buffer, pH 7.0. The rate of binding to serum protein was high (93-96%), but the effect of the addn. of serum on the antibacterial activity was not marked indicating that such binding is reversible. Development of resistance in vitro could be achieved in a similar way with I and cefazolin. There was a stepwise emergence and a slow increase in resistance in Staphylococci and a more rapid one in Escherichia coli. Although I was hydrolyzed by .beta.-lactamases, it was more stable against various crude enzymes than cefazolin and cephalothin.

ST cefazedone antibacterial activity; bactericide cefazedone;

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cephalothin bactericide cefazedone; cefazolin bactericide cefazedone
 IT Clostridium perfringens
 Enterobacter
Escherichia coli
 Klebsiella
Mycobacterium tuberculosis
 Proteus
 Pseudomonas aeruginosa
 Serratia marcescens
 Staphylococcus
 Streptococcus
 (cefazedone **inhibition** of)
 IT 58-71-9 27164-46-1 63521-15-3
 RL: BAC (Biological activity or effector, except adverse); BIOL
 (Biological study)
 (bactericidal activity of)

L94 ANSWER 98 OF 108 HCAPLUS COPYRIGHT 1998 ACS
 AN 1976:84140 HCAPLUS
 DN 84:84140
 TI Tumor regression caused by endotoxins and mycobacterial fractions
 AU Ribí, Edgar E.; Granger, Donald L.; Milner, Kelsey C.; Strain, S.
 Michael
 CS Rocky Mt. Lab., Natl. Inst. Allergy Infect. Dis., Hamilton, Mont.,
 USA
 SO J. Natl. Cancer Inst. (1975), 55(5), 1253-7
 CODEN: JNCIAM
 DT Journal
 LA English
 CC 1-5 (Pharmacodynamics)
 AB Oil drop preps. contg. trehalose mycolate (P3) (isolated from wax D
 from **Mycobacterium tuberculosis** strain Aoyamia
 B) and bacterial endotoxin produced cure rates of up to 90% in
 guinea **pigs** with transplanted hepatocarcinoma.
 Regression was faster than with live bacille Calmette Guerin and
 older tumors could be treated successfully. The most effective
 endotoxins were from rough strains of salmonellae, known as Re
 mutants, which could not synthesize and attach the polysaccharide
 portion of the endotoxin.
 ST endotoxin trehalose mycolate neoplasm inhibition; Salmonella
 endotoxin neoplasm inhibition
 IT Toxins
 RL: BIOL (Biological study)
 (endo, neoplasm inhibition by trehalose mycolate and)
 IT Neoplasm inhibitors
 (endotoxins and trehalose mycolate)
 IT **Escherichia coli**
 Salmonella enteritidis
 Salmonella minnesota
 Salmonella typhimurium
 (endotoxins of, neoplasm **inhibition** by trehalose
 mycolate and)
 IT .alpha.-D-Glucopyranoside, .alpha.-D-glucopyranosyl, esters with
 mycolic acids
 RL: BIOL (Biological study)
 (neoplasm inhibition by endotoxins and)

L94 ANSWER 99 OF 108 MEDLINE DUPLICATE 12
 AN 75074475 MEDLINE
 DN 75074475
 TI [Present applications of **malaria**therapy].
 Applications actuelles de la malariatherapie.
 AU Lupascu G
 SO BULLETIN OF THE WORLD HEALTH ORGANIZATION, (1974) 50 (3-4) 165-7.
 KATHLEEN FULLER BT/LIBRARY 308-4290

Journal code: C80. ISSN: 0042-9686.
 CY Switzerland
 DT Journal; Article; (JOURNAL ARTICLE)
 LA French
 EM 197505
 CT Check Tags: Human

Anopheles
 Antimalarials: TU, therapeutic use
 Drug Resistance
 English Abstract

***Hyperthermia, Induced**

Malaria: DT, drug therapy
 Malaria: TH, therapy
 Malaria: TM, transmission
 Neurosyphilis: DT, drug therapy
 Neurosyphilis: TH, therapy
 Penicillins: TU, therapeutic use
 Plasmodium
 Treponema

L94 ANSWER 100 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.

AN 74119494 EMBASE

TI Malaria in New York City. III. 1940 to 1959.

AU Harvey R.P.; Imperato P.J.; Shookhoff H.B.

CS City New York Dept. Hlth, New York, N.Y., United States

SO N.Y.ST.J.MED., (1973) 73/21 (2601-2605).

CODEN: NYSJAM

LA English

AB A change in the epidemiology of malaria in New York City occurred between 1940 and 1959. The major change was in the source of infection from indigenously acquired cases to imported cases. With this change, new age sex specific attack rates were recognized. Large scale importation of cases failed to produce an endemic outbreak of disease, and in the years between World War II and the Korean War, and the years between the Korean War and the Vietnam War, the virtual disappearance of malaria continued to be observed. In 1959 only 2 cases were reported in the entire city population. Drug addict associated malaria and **malariotherapy** for the treatment of syphilis ceased during the 1940s. The use of quinine for dilution of heroin in New York City undoubtedly played an important role in the former. With the cessation of the Korean conflict, malaria cases became limited to travelers to endemic areas and the infrequent infection resulting from blood transfusion.

CC 005.02.12.00.00.

005.02.13.03.00.

005.02.14.00.00.

005.02.22.02.00.

017.03.07.00.00.

CT EMTAGS: infection (0310); epidemiology (0400); North America (0405); prevention (0165)

Medical Descriptors:

*malaria

*plasmodium vivax

*plasmodium falciparum

L94 ANSWER 101 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1972:535580 HCAPLUS

DN 77:135580

TI Antibacterial activity of pyrimidine and pyrrolo (3,2-d)pyrimidine derivatives

AU Pershin, G. N.; Sherbakova, L. I.; Zykova, T. N.; Sokolova, V. N.

CS Vses. Nauchno-Issled. Khim.-Farm. Inst. im. Ordzhonikidze, Moscow, USSR

SO Farmakol. Toksikol. (Moscow) (1972), 35(4), 466-71

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CODEN: FATOAO
DT Journal
LA Russian
CC 3-2 (Biochemical Interactions)
AB Most of the 85 pyrimidine and pyrrolopyrimidine derivs. studied were bacteriostatic toward **Mycobacterium tuberculosis**, 43 were bacteriostatic toward *Lactobacillus casei*, and none were active against *Escherichia coli*. 6-Chloro-N-[2-(1-cyclohexen-1-yl)ethyl]-5-(2-propen-1-yl)-4-pyrimidinamine (I) [19674-87-4], 7-(butylthio)-2,5-dimethyl-1H-pyrrolo[3,2-d]pyrimidine (II) [36557-26-3], and 6 other compds. bacteriostatic toward **M. tuberculosis**, after administration to tuberculous mice, had no effect on the disease.
ST pyrimidine deriv bacteria inhibition; pyrrolopyrimidine deriv bacteria inhibition; tuberculosis inhibition pyrimidine deriv
IT Bactericides, Disinfectants and Antiseptics
(pyrimidine and pyrrolopyrimidine derivs. as)
IT **Escherichia coli**
Lactobacillus casei
Mycobacterium tuberculosis
(pyrimidine and pyrrolopyrimidine derivs. inhibition of)
IT 5H-Pyrrolo[3,2-d]pyrimidine, derivs.
Pyrimidine, derivs.
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(bactericidal activity of)
IT 19674-87-4 36557-26-3
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(bactericidal activity of)

L94 ANSWER 102 OF 108 BIOSIS COPYRIGHT 1998 BIOSIS
AN 72:190109 BIOSIS
DN BA54:20103
TI THE COURSE OF THE FLUORESCENT ANTIBODY LEVEL DURING HUMAN MALARIA INDUCED BY **MALARIO THERAPY** WITH PLASMODIUM-VIVAX.
AU GARIN J P; AMBROISE-THOMAS P; KIEN TRUONG T; SALIOU P
SO BULL W H O 44 (5). 1971 689-699. CODEN: BWHOA6 ISSN: 0366-4996
LA Unavailable
ST IMMUNO GLOBULINS
CC Biochemical Studies-Proteins, Peptides and Amino Acids 10064
Movement 12100
Pathology, General and Miscellaneous-Diagnostic 12504
Pathology, General and Miscellaneous-Therapy *12512
Metabolism-Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies 15002
Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies 15004
Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Immunology and Immunochemistry-General; Methods 34502
Immunology, Parasitological *35000
Medical and Clinical Microbiology-Serodiagnosis *36504
Parasitology-Medical *60504
BC Sporozoa 35400
Hominidae 86215

L94 ANSWER 103 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1971:84343 HCAPLUS
DN 74:84343
TI Anti-bacterial activity of unguic acid
AU Leikola, Erkki; Teppo, Anna M.; Vilppula, H.
CS Res. Dep., Orion-Yhtymä Oy, Helsinki, Finland
KATHLEEN FULLER BT/LIBRARY 308-4290

SO Ann. Med. Exp. Biol. Fenn. (1970), 48(4), 234-7
CODEN: AMEBA7
DT Journal
LA English
CC 8 (Microbial Biochemistry)
AB Ungulic acid inhibited the growth of Streptococcus faecalis and Staphylococcus aureus in concns. of 1.6-2.3mM, while Pseudomonas aeruginosa, Kbsiella pneumoniae, and Proteus mirabilis were inhibited by concns. of 7.8mM. Ungulic acid did not **inhibit Escherichia coli**. Ungulic acid also had bacteriostatic activity against **Mycobacterium tuberculosis**. The min. inhibitory concn. of ungulic acid in vitro was compared to the concn. of ungulic acid in normal **human** epidermis.
ST ungulic acid antibacterial activity; antibacterial activity ungulic acid
IT Antibiotics, biological studies
(from animals, ungulic acid as)
IT Ungulic acid
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(bactericidal activity of)

L94 ANSWER 104 OF 108 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 67-06204H [01] WPIDS
CR 66-13596F [00]
TI 2-Substd. 5-nitrofurans antibiotics.
DC B03 C02
PA (PHAA) PHARMACIA AB
CYC 2
PI CA 811726 A (6801)*
NL 138126 B (7310)
PRAI SE 63-2193 630228; SE 64-1845 640215
AB CA 811726 A UPAB: 930831
Cpds. tautomeric forms and acid addition salts. R1, R2, R3 and R4 = H, alkyl (one or two only) or -COR6 (one only) where R6 = H or (1-3C) alkyl opt. substd. with halogenR5 = H, or may form a double bonds with R1, R2 or R4
Antibiotics.
Shown to be effective against **M. tuberculosis**,
Staphylococcus aureus, **E. coli**, Salmonella and Shigella. **Mice**,
treated orally with 50 mg./kg. body wt., were still excreting active cpds. in urine, up to 6 hrs. after treatment
3-amino-4-methyl-5-(5-nitro-2-furyl)-1:2:4-triazole
FS CPI
FA AB
MC CPI: C07-A01; C07-D13; C12-A01; C12-A04

L94 ANSWER 105 OF 108 MEDLINE
AN 69064310 MEDLINE
DN 69064310
TI [A new technic to control **malaria**therapy in syphilogenic psychoses].
Uma nova tecnica de controle da malarioterapia nas psicoses sifilogenicas.
AU Garcia J A; Silva J R; Lopes P F
SO REVISTA BRASILEIRA DE MEDICINA, (1967 Nov) 24 (11) 902-6.
Journal code: RJ5. ISSN: 0034-7264.
CY Brazil
DT Journal; Article; (JOURNAL ARTICLE)
LA Portuguese

EM 196903
CT Check Tags: Human
Brazil
*Delirium, Dementia, Amnestic, Cognitive Disorders: ET, etiology
***Hyperthermia, Induced**
Penicillins: TU, therapeutic use
Plasmodium: IM, immunology
*Syphilis: CO, complications
Syphilis: EP, epidemiology
United States

L94 ANSWER 106 OF 108 HCAPLUS COPYRIGHT 1998 ACS
AN 1967:114128 HCAPLUS
DN 66:114128
TI Specificity of resistance to tuberculosis and to salmonellosis
stimulated in **mice** by oil-treated cell walls
AU Ribb, Edgar; Brehmer, Werner; Milner, Kelsey C.
CS Natl. Insts. of Health, Rocky Mt. Lab., Hamilton, Mont., USA
SO Proc. Soc. Exp. Biol. Med. (1967), 124(2), 408-13
CODEN: PSEBAA
DT Journal
LA English
CC 13 (Immunochimistry)
AB When **mice** were vaccinated s.c. with untreated or mineral
oil (0.48 ml./100 mg. cell wall)treated preps. of dried cell walls
(0.4-10 .mu.g.) from Salmonella enteritidis and challenged 14 days
later with viable S. enteritidis (1 .times. 10⁷ plate count units,
i.p.), they were protected 4 days after challenge in a dose-graded
response; 90% of the unvaccinated **mice** died. **Mice**
receiving endotoxin (0.5-50 .mu.g.) from Citrobacter [Escherichia]
freundii were not significantly protected. **Mice** given
oil-treated and nontreated **E. coli**
cell wall preps. (0.4-100 .mu.g., i.v.) and challenged i.p. 24 hrs.
later with 8 .times. 10⁷ cells of S. typhosa were protected; 75-100%
of the controls died within 3 days. The cell walls of BCG,
oil-treated or not, were not protective. I.p. vaccinations of
oil-treated cell wall preps. from S. typhimurium (100 .mu.g.) and
Brucella abortus (1000 .mu.g.) protected **mice** more against
i.v. challenge 24 hrs. later with 200 .times. 10⁶
Mycobacterium tuberculosis H37RV cells than the
oil-treated cell wall preps. from Listeria monocytogenes (1000
.mu.g.) and BCG (500 .mu.g.), and the protection correlated roughly
with the endotoxin content. Oil-treated cell wall preps. (100-1000
.mu.g.) from S. typhimurium, B. abortus, **M.**
tuberculosis H37RV, L. monocytogenes, and BCG increased the
survival time in **mice** challenged i.v. with 4 .times. 10⁷
cells of H37RV 30 days after the i.p. vaccination, and the BCG cell
wall prepn. was at least as effective as the endotoxin-contg.
vaccines, and much more so than the cell walls of L. monocytogenes.
Mice treated i.v. with oil-treated cell walls (100-500
.mu.g.) from BCG and challenged 4 weeks later with virulent tubercle
bacilli by aerosol were protected, while none were protected when
given oil-treated cell walls (100-1000 .mu.g.) from S. typhimurium,
B. abortus, or L. monocytogenes. Coating with oil, which was
previously reported (CA 65, 20659d) to be essential to render cell
walls of BCG protective to **mice** against challenge with
tubercle bacilli by aerosol, does not affect the specificity of
reactions conditioned by cell walls in this and other systems. 17
references.

ST VACCINES CELL WALLS; CELL WALLS VACCINES; MINERAL OIL ANTIGENS;
ANTIGENS MINERAL OIL; OIL MINERAL ANTIGENS; BACTERIAL PATHOGENS OIL;
PATHOGENS BACTERIAL OIL
IT Brucella
(abortus, tuberculosis resistance after injection of oil-treated
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cell walls of)

IT Listeria
(monocytogenes, tuberculosis resistance after injection of
oil-treated cell walls of)

IT Salmonella
(typhi and typhimurium, vaccine for, oil-treated cell walls as)

IT Tuberculosis
(vaccine for, oil-treated cell walls as)

L94 ANSWER 107 OF 108 HCAPLUS COPYRIGHT 1998 ACS

AN 1968:76737 HCAPLUS

DN 68:76737

TI In vitro and in vivo chemotherapeutic properties of the antibiotic
myxin

AU Grunberg, Emanuel; Berger, Julius; Beskid, George; Cleeland, Roy;
Prince, Herbert N.; Titsworth, Edith

CS Hoffmann-La Roche Inc., Nutley, N. J., USA

SO Chemotherapia (1967), 12(5), 272-81
CODEN: CMTRAG

DT Journal

LA English

CC 15 (Pharmacodynamics)

AB Myxin (6-methoxy-1-phenazinol 5,10-dioxide) (I) is an antibiotic
that displays a broad in vitro spectrum including activity against
gram-pos. and gram-neg. bacteria, **Mycobacterium
tuberculosis**, Mycoplasma gallinarum, Candida albicans,
filamentous fungi, dermatophytes, helminths, and protozoa. The in
vitro antibacterial effect could be partially overcome by the addn.
of cysteine or Na thioglycolate to the growth medium. I was
cytotoxic for **monkey** kidney cells. I was not absorbed
when administered by the oral or s.c. routes to **mice**. I
was active when administered i.p. to **mice** infected
systematically with Streptococcus pyogenes, Diplococcus pneumoniae,
Staphylococcus aureus, Escherichia coli, and Neisseria meningitidis
as well as against **mice** implanted with sarcoma 180, but
was without effect when tested by this same route against fungi,
viruses, and Ehrlich carcinoma. When tested for local
chemotherapeutic effects against s.c. bacterial infections, I
exerted marked activity against Streptococcus pyogenes, S. aureus,
and Proteus vulgaris, moderate activity against E. coli, and a
slight effect in the case of Pseudomonas aeruginosa. The antibiotic
also exerted a marked effect against a s.c. Trichomonas vaginalis
infection in **mice** when administered by infiltration as
well as a slight effect against the s.c. C. albicans infection in a
similar exptl. model. I administered orally showed slight to
moderate anthelmintic activity against Syphacia obvelata and
Hymenolepis nana.

ST MYXIN ACTION SPECTRUM; TRICHOMONAS MYXIN; CANDIDA MYXIN; PROTOZOA
MYXIN; BACTERIA MYXIN; DERMATOPHYTES MYXIN; HELMINTHS MYXIN; FUNGI
MYXIN

IT Staphylococcus
(aureus, infection with, myxin in treatment of)

IT **Escherichia coli**
(infection with, myxin in **treatment** of)

IT Neisseria
(meningitidis, infection with, myxin in treatment of)

IT Anthelmintics
Neoplasm inhibitors
Antibiotics, biological studies
(myxin as)

IT Diplococcus
(pneumoniae, infection with, myxin in treatment of)

IT Streptococcus
(pyogenes, infection with, myxin in treatment of)

IT Trichomonas
(vaginalis, infection with, myxin in treatment of)
IT Proteus
(vulgaris, infection with, myxin in treatment of)
IT 13925-12-7
RL: BAC (Biological activity or effector, except adverse); BIOL
(Biological study)
(antibiotic activity of)
IT 52-90-4, biological studies
RL: BAC (Biological activity or effector, except adverse); BIOL
(Biological study)
(inhibition by antibiotic activity of myxin by)

L94 ANSWER 108 OF 108 MEDLINE
AN 68049720 MEDLINE
DN 68049720
TI [Notes on the practice of **malariotherapy**].
Note sulla pratica della malarioterapia.
AU Marotta G
SO RIVISTA DI MALARIOLOGIA, (1967 Jun) 46 (1) 23-36.
Journal code: TN5.
CY Italy
DT Journal; Article; (JOURNAL ARTICLE)
LA Italian
EM 196802
CT Check Tags: Human
Antimalarials: TU, therapeutic use
Arteriosclerosis: TH, therapy
***Hyperthermia, Induced**
Hyperthermia, Induced: AE, adverse effects
Malaria: DT, drug therapy
***Neurosyphilis: TH, therapy**
Paralysis: TH, therapy
Thromboangiitis Obliterans: TH, therapy
Vascular Diseases: TH, therapy

patients well tolerated the non-invasive WBH as well as the high dose BC supplementation. Apart from one patient who died after 4 months, all the others underwent an HIV burden diminution, clinical improvement and amelioration of laboratory data, along with an subjective improvement of their life quality. With reference to control groups, namely (a) only WBH applied with extracorporeal procedure to 31 AIDS patients, and (b) only BC supplementation at high dosage applied to 64 ARC patients, the combined physical and BC supplemental treatments clearly showed a better and longer lasting response.

CT Check Tags: Female; Human; Male
Acquired Immunodeficiency Syndrome: DT, drug therapy
***Acquired Immunodeficiency Syndrome: TH, therapy**
 Adult
 Antioxidants: TU, therapeutic use
 AIDS-Related Complex: TH, therapy
 *Carotene: TU, therapeutic use
 *Food, Fortified
***Hyperthermia, Induced**
 RN 36-88-4 (Carotene); 7235-40-7 (Beta Carotene)
 CN 0 (Antioxidants)

L94 ANSWER 35 OF 108 MEDLINE
 AN 95396279 MEDLINE
 DN 95396279
 TI Hyperthermic therapy for HIV infection.
 AU Owens S D; Gasper P W
 CS Department of Pathology, College of Veterinary and Biomedical Sciences, Colorado State University, Ft Collins 80523, USA.
 SO MEDICAL HYPOTHESES, (1995 Apr) 44 (4) 235-42. Ref: 57
 Journal code: MOM. ISSN: 0306-9877.
 CY ENGLAND: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LA English
 FS Priority Journals
 EM 199512
 AB The objective of this paper is to review what is known about the antiviral effects of fever and to highlight the scientific evidence supporting the hypothesis that hyperthermic therapy may prove to be a beneficial treatment modality for persons infected with HIV. Our hyperthermic hypothesis is based upon the mutant escape, quasispecies theory of HIV antigenic diversity. We propose that, if initiated during the asymptomatic stage of HIV infection, hyperthermia may prove to decrease the number of mutant HIV strains arising due to evolutionary pressures created by the patient's immune system, with a resultant prolongation of the asymptomatic period of infection. A review of the literature from three areas of investigation: the immune response to fever, heat as a tumor killing agent, and preliminary studies with fever and retroviral infections, strongly suggests that there is a good scientific basis for the use of hyperthermic therapy in a multimodal treatment approach to HIV infection.

CT Check Tags: Animal; Human; Support, Non-U.S. Gov't
Acquired Immunodeficiency Syndrome: PP, physiopathology
***Acquired Immunodeficiency Syndrome: TH, therapy**
 Evolution
 *Fever: PP, physiopathology
***Hyperthermia, Induced**
 HIV: GD, growth & development
 *HIV: PH, physiology
 HIV: PY, pathogenicity
 HIV Infections: PP, physiopathology

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*HIV Infections: TH, therapy
 Models, Biological
 Neoplasms: TH, therapy
 Neoplasms, Experimental: TH, therapy
 Retroviridae Infections: PP, physiopathology
 Retroviridae Infections: TH, therapy

L94 ANSWER 36 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.

AN 95098290 EMBASE

TI Anaemia and **Plasmodium falciparum** infections
 among young children in an holoendemic area, Bagamoyo, Tanzania.

AU Premji Z.; Hamisi Y.; Shiff C.; Minjas J.; Lubega P.; Makwaya C.

CS Bagamoyo Bed Net Project, PO Box 65011, Dar es Salaam, Tanzania,
 United Republic of

SO Acta Tropica, (1995) 59/1 (55-64).

ISSN: 0001-706X CODEN: ACTRAQ

CY Netherlands

DT Journal

FS 004 Microbiology

007 Pediatrics and Pediatric Surgery

017 Public Health, Social Medicine and Epidemiology

LA English

SL English

AB Although the aetiology of anaemia in tropical areas is multifactorial, **Plasmodium falciparum** malaria is commonly associated with anaemia in children living in holoendemic malaria areas. Such an association was examined in a population based study of 338 children 6 to 40 months of age living in the Bagamoyo area of Tanzania. Stepwise regression analysis showed that fever and **parasitaemia** were effective in predicting anaemia and that the anaemic condition was age dependent. The majority of the children were iron deficient, followed by normochromic macrocytic anaemias. There was strong evidence in this age group that the anaemia was associated with malaria and not geohelminth infection. The importance of malaria and anaemia as a cause of childhood morbidity in Africa is discussed. This condition has taken on new significance with the realization that blood transfusions commonly used to **treat** severe anaemia are a major vehicle for Human **Immunodeficiency** Virus (**HIV**) transmission.

CT EMTAGS: **etiology** (0135); **epidemiology** (0400);
 invertebrate (0723); protozoon (0751); infection (0310); therapy
 (0160); africa (0403); africa south of the sahara (4032); mammal
 (0738); **human** (0888); major clinical study (0150); infant
 (0014); child (0022); article (0060)

Medical Descriptors:

*anemia: ET, etiology

*anemia: EP, epidemiology

***plasmodium falciparum**

*malaria falciparum: EP, epidemiology

*childhood disease: ET, etiology

*childhood disease: EP, epidemiology

*blood transfusion

population research

tanzania

morbidity

africa

human

major clinical study

infant

child

article

L94 ANSWER 37 OF 108 MEDLINE

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AN 96092654 MEDLINE
 DN 96092654
 TI Mechanism of the effect of thermotherapy as applied to AIDS.
 AU Moreira M B
 SO MEDICAL HYPOTHESES, (1995 Jul) 45 (1) 5-6.
 Journal code: MOM. ISSN: 0306-9877.
 CY ENGLAND: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199603
 AB Artificially induced thermal intermittence using thermogenic agents was utilized to treat AIDS patients in an attempt to make an analogy with the sterilization process by tyndallization employed in laboratories. It is known that micro-organisms are more sensitive to discontinuous than to constant heat. The author believes that the AIDS virus may be either destroyed or weakened using this method which may also provoke an immune stimulus over the body's system of defense, especially over the bone marrow, with the consequent increase of the indexes of lymphocins, opsonins and hematogenesis.
 CT Check Tags: Comparative Study; Human
 *Acquired Immunodeficiency Syndrome: TH, therapy
 Heat
 *Hyperthermia, Induced
 Neoplasms: TH, therapy
 Sterilization: MT, methods

 L94 ANSWER 38 OF 108 HCAPLUS COPYRIGHT 1998 ACS
 AN 1995:309101 HCAPLUS
 DN 122:64331
 TI Method for treating neurological disorders using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space
 IN Kim, Sinil; Howell, Stephen B.
 PA Depotech Corp., USA
 SO PCT Int. Appl., 43 pp.
 CODEN: PIXXD2
 PI WO 9426250 A1 941124
 DS W: CA, JP
 AI WO 93-US4645 930514
 DT Patent
 LA English
 IC ICM A61K009-127
 CC 63-5 (Pharmaceuticals)
 AB A method is disclosed for ameliorating a neurol. disorder (tumor, virus infection, etc.) in a **human** by administration to the cerebrospinal fluid (CSF) of a therapeutic agent in a dispersion system which allows the therapeutic agent to persist in the cerebro-ventricular space. Prodn. of a synthetic membrane vesicle having multiple nonconcentric chambers contg. ara-C which are bounded by a single bilayer membrane is described. The ara-C prepn. was used in intrathecal and intraventricular treatment with patients having histol. proven cancer and evidence of neoplastic meningitis. Pharmacokinetic data, toxicity data, and cytol. response are included.
 ST neurol disorder therapeutic dispersion cerebrospinal fluid; tumor neurol therapeutic dispersion cerebrospinal fluid; cerebroventricular space cerebrospinal fluid neurol therapeutic
 IT Polymers, biological studies
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (matrix; neurol. disorder treatment using administration to cerebrospinal fluid with therapeutic dispersion allowing persistence in cerebro-ventricular space)
 IT Bactericides, Disinfectants, and Antiseptics
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Enterobacter
Escherichia coli
 Haemophilus influenzae
 Klebsiella
 Listeria monocytogenes
Mycobacterium tuberculosis
 Neisseria meningitidis
 Proteus (bacterium)
 Pseudomonas aeruginosa
 Staphylococcus aureus
 Streptococcus pneumoniae
 (neurol. bacteria infection **treatment** using
 administration to cerebrospinal fluid with therapeutic dispersion
 allowing persistence in cerebro-ventricular space)
 IT Cell cycle
 (neurol. disorder treatment using administration to cerebrospinal
 fluid with cell cycle phase-specific therapeutic dispersion
 allowing persistence in cerebro-ventricular space)
 IT Anti-infective agents
 Autoimmune disease
 Cerebrospinal fluid
 Eukaryote
 Neoplasm inhibitors
 Nervous system agents
 Prokaryote
 (neurol. disorder treatment using administration to cerebrospinal
 fluid with therapeutic dispersion allowing persistence in
 cerebro-ventricular space)
 IT Antibodies
 Glycolipids
 Carbohydrates and Sugars, biological studies
 Proteins, biological studies
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (neurol. disorder treatment using administration to cerebrospinal
 fluid with therapeutic dispersion allowing persistence in
 cerebro-ventricular space)
 IT Blastomyces
 Candida
 Coccidioides immitis
 Cryptococcus (fungus)
 Fungicides and Fungistats
 Histoplasma
 Nocardia
 (neurol. fungus infection treatment using administration to
 cerebrospinal fluid with therapeutic dispersion allowing
 persistence in cerebro-ventricular space)
 IT Metabolism
 (neurol. metabolic dysfunction treatment using administration to
 cerebrospinal fluid with therapeutic dispersion allowing
 persistence in cerebro-ventricular space)
 IT Virucides and Virustats
 (neurol. virus infection treatment using administration to
 cerebrospinal fluid with therapeutic dispersion allowing
 persistence in cerebro-ventricular space)
 IT Drug interactions
 (oral dexamethasone redn. of toxicity of ara-C dispersion
 intrathecal and intraventricular treatment in cancer patients
 with neoplastic meningitis)
 IT Membranes
 (synthetic, vesicles; neurol. disorder treatment using
 administration to cerebrospinal fluid with therapeutic dispersion
 allowing persistence in cerebro-ventricular space)
 IT Interphase, biological
 (S-phase, neurol. disorder treatment using administration to

LA German
 EM 198907
 CT Check Tags: Female; Human; Male
 ***Acquired Immunodeficiency Syndrome: CO, complications**
 Adult
 *Anus Diseases: CO, complications
 Combined Modality Therapy
 Condylomata Acuminata: CO, complications
 Condylomata Acuminata: SU, surgery
 Diathermy
 English Abstract
 Hemorrhoids: CO, complications
 Hemorrhoids: TH, therapy
 Middle Age
 *Rectal Diseases: CO, complications
 Rectal Fistula: CO, complications
 Rectal Fistula: TH, therapy

L94 ANSWER 82 OF 108 MEDLINE
 AN 89096588 MEDLINE
 DN 89096588
 TI An approach to AIDS therapy using hyperthermia and membrane modification.
 AU Yatvin M B
 CS University of Wisconsin Medical School, Madison 53706.
 SO MEDICAL HYPOTHESES, (1988 Nov) 27 (3) 163-5. Ref: 31
 Journal code: MOM. ISSN: 0306-9877.
 CY ENGLAND: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LA English
 FS Priority Journals
 EM 198904
 AB Altering the biophysical characteristics of cell membranes by diet and membrane perturbing agents markedly influences thermosensitivity of cells. Likewise, manipulation of viral envelopes either by altering their lipid composition by diet or by the use of agents that perturb the lipid envelope influence infectivity of enveloped viruses and the progression of viral disease. The use of hyperthermia and envelope modification as a combined approach to treat AIDS has until now neither been suggested nor attempted. On the basis of my previous work and a review of the literature, I theorize that the combination of hyperthermia with procedures designed to alter the viral envelope will likely result in an increased viral sensitivity and be useful clinically for treatment of patients with enveloped viral diseases such as AIDS.

CT Check Tags: Human
 ***Acquired Immunodeficiency Syndrome: TH, therapy**
 Butylated Hydroxytoluene: TU, therapeutic use
 ***Hyperthermia, Induced**
 HIV: ME, metabolism
 Membrane Fluidity
 Membrane Lipids: ME, metabolism
 RN 128-37-0 (Butylated Hydroxytoluene)
 CN 0 (Membrane Lipids)

L94 ANSWER 83 OF 108 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
 AN 87135493 EMBASE
 TI Use of pyrimethamine-sulfadoxine (Fansidar) in prophylaxis against chloroquine-resistant **plasmodium falciparum** and **Pneumocystis carinii**.
 AU Pearson R.D.; Hewlett E.L.
 CS Division of Geographic Medicine, Department of Medicine, University
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